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PERICARDIOSTOMY FOR SUPPURATIVE PERICARDITIS*

A Report Concerning Ten New Cases and Twenty-Eight Cases from the Literature

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DURING the last half century, there have been numerous contributions to the literature dealing with suppurative pericarditis. In recent years comprehensive reviews by Shipley and Winslow,[1] Bisgard,[2] Truesdale[3] and others have summarized the present knowledge of the subject and have collected the recorded cases treated by pericardiostomy. Thus, up to January 1, 1934, Shipley and Winslow[1] were able to collect 227 cases. In this series, there were 102 deaths, a mortality of 44 per cent.

In this paper, we report 5 cases of acute suppurative pericarditis in which pericardiostomy was performed by one of us (J. W. S.), record 5 additional cases from the records of the Boston City Hospital, and review a series of 28 cases collected from the literature from January 1, 1934, to January 1, 1940. The addition of these cases from the Boston City Hospital brings the total of the recorded cases to 265. The 10 reported cases are summarized in Table 1, and the 28 collected cases in Table 2.

The frequency of the disease is emphasized by Pyrah and Pain,[27] who found acute pericarditis in 214, or 2.7 per cent, of 7965 autopsies performed at the Leeds General Infirmary from 1921 to 1931. Of these, 91, or 1.1 per cent of all cases, were classified as acute suppurative. This figure is supported by an incidence of 177 cases, or 1.8 per cent, described as acute suppurative among 9536 autopsies performed at the Boston City Hospital from 1916 through 1938. In the same trend is a figure of 1.1 per cent of 1795 autopsies at the Massachusetts Memorial Hospitals. Stenbuck[12] states that in the nine years prior to December, 1936, there were 4000 autopsies at the Mt. Sinai Hospital. In this series, there were 67 cases of acute purulent pericarditis, in which only 1 patient had been operated on. Many of the cases in this series were, of course, a single manifestation of an overwhelming sepsis and, as such, were but an incident in the progress of the fatal illness.

Notwithstanding the frequency of its occurrence, Cabot[28] states that 77 per cent of all cases of pericarditis found at autopsy at the Massachusetts General Hospital were not diagnosed during the life of the patient. These figures, however, are somewhat misleading because, although these cases are termed acute pericarditis, they were not all purulent. Thus, it seems to us that cases amenable to surgery might have a higher percentage of correct diagnoses, since, as mentioned above, many cases termed acute pericarditis are incidental and, indeed, terminal.

This impression is confirmed by Bigger[29] in analyzing 17 cases of acute fibrinopurulent or purulent pericarditis from the Medical College of Virginia Hospitals. Seven of the frankly purulent ones were correctly diagnosed, and 6 were drained. Of the remaining 10, first diagnosed at autopsy, only 4 had a demonstrable increase in pericardial fluid, and in the other 6, death occurred from an overwhelming infection before there had been an opportunity for pus to form in the pericardial sac. All 4 of the patients who were found on post-mortem examination to have a demonstrable increase in fluid were moribund when admitted to the hospital, 3 dying in less than twelve hours and 1 in twenty-four hours.

Since the disease is never primary, except in the sense that it occurs as a complication of direct trauma (although Morin[4] has reported a case that

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TABLE 1. *Data on Reported Cases.*

CASE NO.	DATE	AGE	SEX	ORGANISMS	DURATION OF ILLNESS	ANTECEDENT DISEASE	PHYSICAL SIGNS	X-RAY	TAP	TYPE OF OPERATION	DRAINAGE MATERIAL	IRRIGATION	RESULT
		yr.											
1	1937	48	M	Type 8 pneumococcus	4 days	Pneumonia (left lung)	Diagnostic (rub)	Diagnostic	+	Resection left 4th and 5th cartilages	None	Saline solution	Died, 10 days (septicemia)
2	1938	13	F	*Staph. aureus* (hemolytic)	12 hours	Pneumonia, septicemia	Diagnostic (rub)	Not diagnostic	+	Resection left 4th and 5th cartilages	None	Saline solution	Died, 42 hours (septicemia)
3	1939	33	M	*Staph. aureus*	4 days	Stab wound	Diagnostic (no rub)	Diagnostic	Not done	Resection left 5th and 6th cartilages	None	Dakin's solution	Recovered
4	1940	44	F	*Staph. albus*	2 days	Ulcerative colitis	Diagnostic (rub)	Not diagnostic	+	Resection left 5th and 6th cartilages	None	Dakin's solution	Died, 7 days (septicemia)
5	1940	15	M	Type 5 pneumococcus	Few hours	Lobar pneumonia (left lung)	Diagnostic (rub)	Diagnostic	No fluid obtained	Resection left 5th and 6th cartilages	None	Dakin's solution	Recovered
6	1913	30	M	Pneumococcus	Few hours	Pneumonia (right lung)	Diagnostic (rub)	Diagnostic	+	Resection right 4th and 5th cartilages	Cigarette	None	Recovered
7	1917	17	M	?	28 days	Bilateral empyema	Diagnostic (no rub)	Diagnostic	+	Resection left 5th cartilage	Rubber tissue	None	Died, 28 days (pneumonia)
8	1921	14	F	*Staph. aureus*	1 day	Septicemia	Diagnostic (no rub)	Diagnostic	+	Resection left 5th and 6th cartilages	Rubber tube and gauze	None	Died, 23 days (pulmonary edema)
9	1929	18	M	*Staph. aureus*	2 days	Broncho-pneumonia	Diagnostic (no rub)	Diagnostic	+	Resection left 6th and 7th cartilages	Rubber tube	Saline solution	Recovered
10	1930	12	M	Type 1 pneumococcus	Few hours	Lobar pneumonia and empyema (right lung)	Diagnostic (no rub)	Not diagnostic	+	Resection of one cartilage (closed drainage)	Rubber tube	Dakin's amniotic fluid	Died, 15 days

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he considers primary), pericarditis may well be obscured by the original infection, particularly, as is so often true, if this infection is within the thorax. Thus, in our 10 cases, 7 were secondary to pneumonia or empyema, or both; 2 were secondary to septicemia, and 1 followed a stab wound. Twenty-seven of the 58 cases reported since 1934 followed affections of the chest, as shown in Table 3. Although it is not the purpose of this paper to go exhaustively into diagnosis, one cannot but assume, in the light of the foregoing, that an improvement in diagnosis will be obtained only if the physician, in the absence of localizing symptoms, makes careful repeated examinations of the cardiac region. Further, as Bigger[23] points out, if a friction rub is not heard and there is no appreciable increase in the amount of intrapericardial fluid, the diagnosis cannot be made. No rub was heard in 5 of our 10 cases. The roentgenographic and fluoroscopic findings may, therefore, be of no diagnostic aid in a certain percentage of cases, and this was true in 2 of the 10 cases that we report. Nevertheless, roentgenographic examination is of the utmost importance in diagnosis and should be carried out in the upright and prone positions, as suggested by Hodges,[29] so that alterations in the pericardial shadows resultant on these changes in position may be observed.

Heyl[9] has described fluid waves moving down the borders of the pericardial shadow, initiated by systole, that he observed fluoroscopically and that he considered pathognomonic of fluid in the pericardium. Not infrequently, the patient is too ill to permit careful fluoroscopy.

In cases of questionable diagnosis, one may have recourse to pericardicentesis or exploratory operation. Although the former procedure carries undeniable risks (we have seen unwitting puncture of an enlarged heart result in death from hemopericardium with cardiac tamponade), we believe it should be carried out before exploration is undertaken. It was performed without incident in 9 of our 10 cases, and, in the case in which it was not performed (Case 3), pus draining from the stab wound established the diagnosis.

On the basis of our personal experience and the recorded cases, we cannot agree with Elkin,[30] who believes it is safer to expose the pericardium by operation than to perform pericardicentesis. Although we agree that pericardiostomy is, itself, attended by little or no risk, we should qualify this by stating that, if fluid is not obtained by pericardicentesis after one or two attempts, further efforts should be abandoned in favor of exploration, particularly if the patient is suffering from the effects of cardiac tamponade.

It is well known that post-mortem examination of patients dying of pneumonia and its complications may reveal a varying accumulation of sterile fluid in the pericardium while there is, at the same time, actual pus in one or both pleural cavities. This has been particularly likely to occur since the advent of chemotherapy. Finland[31] offers the suggestion that the diffusion of the drug into the pericardial fluid suffices to kill such organisms as traverse the pleura *and* pericardium, whereas the initial or continuing infection of the pleura cannot be taken care of because of a low concentration of the drug, the great number and virulence of the organisms or an excessive amount of so-called "inhibiting substance" in the infected pleural cavity. All three of these factors may operate simultaneously.

This chain of circumstances, when present in a patient who has clinical evidence of acute pericarditis, may pose a nice question of judgment, and was actually encountered in 2 of our patients (Cases 5 and 9). Thus, if organisms cannot be demonstrated in the pericardial fluid obtained by pericardicentesis, and the patient has an empyema, or has had pneumonia, and is clinically suffering from the effects of pericarditis, should pericardiostomy be undertaken?

Of course, one cannot answer this question categorically. We believe that there are sound reasons for proceeding with operation in the face of this negative evidence, because of the considerable advantages to be derived from drainage before the pus becomes thick and fibrinous. We have found this to be true in 3 cases in which the patients were operated on before thick pus had formed (Cases 4, 5 and 9); all recovered. Certainly, one cannot know whether the fluid will remain sterile or become purulent, but if the patient is relieved by pericardicentesis and the fluid remains clear, one is, perhaps, justified in repeating the aspiration and observing the character of the fluid for a day or two longer.

Treatment

Although it has been stated that suppurative pericarditis, unoperated, has a mortality of 100 per cent, this is not strictly true, since cases of spontaneous evacuation, with recovery, and of recovery after repeated pericardicenteses can be found in the literature. The percentage of such cases, of course, must be extremely small, and it is to be regretted that, with the advent of the use of sulfanilamide and related compounds, probably increasing numbers of ill-advised trials of such nonoperative measures will be made. It is only the occasional patient with empyema thoracis who recovers with aspiration alone or in combination

TABLE 2. *Data on Collected Cases.*

CASE NO.	AUTHOR	SEX	AGE yr.	PRE-EXISTING DISEASE	ORGANISM	OPERATION	RESULT	COMMENT
11	Morin[4]	M	18	None	Staphylococcus	Pericardiotomy	Died, 2½ months after onset of disease	Autopsy revealed no cause of infection. Authors believe this is a case of "primary pericarditis."
12	Sennels[5]	M	16	Infected left knee	?	Resection of ensiform	Died, 10 days after operation	Multiple metastatic abscesses
13	Gertzenstein and Kreutzer[6]	M	7	Infected right knee	?	Pericardiotomy	Died, 10 days after operation	Autopsy: "bacillary arthritis" (right knee)
14	del Carril, Pflaum and Dechessarri[7]	M	1¼	Pneumonia and empyema	Pneumococcus	Resection of 5th left cartilage	Died, 12 days after operation	
15	Moore[8]	M	12	Pneumonia and empyema	Hemolytic streptococcus	Posterior resection, 5th and 6th ribs	Recovered	Pleural cavity obliterated by previous empyema
16	Heyl[9]	F	1½	Pneumonia (right lung)	Type 4 pneumococcus	Resection left 3rd and 4th cartilages	Recovered	Measles previous to pneumonia
17	Piasecki[10]	F	8	Pneumonia and empyema (left)	Pneumococcus	Resection left 5th cartilage	Recovered	
18		M	10	Pneumonia and empyema (left)	Pneumococcus	Resection left 5th cartilage	Recovered	
19	Wilensky and Lilienthal[11]	F	6	Septicemia and empyema (right)	Nonhemolytic streptococcus	Resection right 5th and 6th cartilages	Recovered	Approached on right side because of right empyema
20	Stenbuck[12]	M	5	Bilateral empyema	Type 2 pneumococcus	Resection left 4th and 5th cartilages	Recovered	Drainage of right empyema and of left pyopneumothorax, with bronchopleural fistula and tension symptoms
21	Bigger[13]	M	14	Bronchogenic carcinoma	*Staph. aureus*	Resection left 5th and 6th cartilages	Died, 21 days after operation	Previous pneumonectomy
22		M	12	Septic arthritis and septicemia	*Staph. aureus*	Resection left 4th and 5th cartilages	Died, 3 days after operation	
23		F	10	Pneumonia and empyema (right)	Nonhemolytic streptococcus	Resection left 4th and 5th cartilages	Recovered	Empyema (right) drained by closed method, 6 days after pericardiostomy
24		M	16	Tuberculous pericarditis, with secondary infection	Nonhemolytic streptococcus	Resection left 5th and 6th cartilages	Died, 4 months after operation	One month before death, had lysis of adhesions about inferior vena cava
25		M	18	Pneumonia and empyema (right)	Pneumococcus	Resection left 4th and 5th cartilages	Recovered	Infection of cartilages, which prolonged convalescence
26	Sicard[14]	F	12	Pneumonia (right)	Pneumococcus	Epigastric approach (Allingham)	Recovered	Author impressed by the epigastric approach
27	Hallock[15]	M	21	Pneumonia (left)	Type 1 pneumococcus	Through 4th left intercostal space	Recovered	
28	Lilienthal[16]	M	54	Calcified pericardium	*Staph. albus*	Sternum split as for pericardiectomy	Died, 2 weeks after operation	
29	Heidenreich, del Sel and Echevarria[17]	M	30	Pneumonia and empyema	Pneumococcus	Type not stated	Died, 3 days after operation	
30	Ortiz Lagunes[18]	M	35	? Pneumonia	"Of the nature of colon bacillus"	Resection left 6th cartilage	Still in hospital and draining	
31	Rayburn[19]	M	2	Laryngeal diphtheria and empyema	Not stated	Partial resection left 5th and 6th cartilages	Died, 3 days after operation	
32	Attix[20]	M	55	Pneumonia (left)	Pneumococcus	Partial resection left 5th, 6th and 7th cartilages	Recovered	
33	Train[21]	M	7	Septicemia	*Staph. aureus*	Epigastric approach (subcostal, in two stages)	Recovered	Multiple soft-tissue and bone abscesses
34	Ross[22]	M	5	? "Influenza"	? *Haemophilus pertussis*	Epigastric approach (subcostal, in two stages)	Recovered	
35	Bigger[23]	M	28	Pneumonia	Type 8 pneumococcus	Resection left 4th and 5th cartilages	Recovered	
36	Guiou[24]	M	9	Septicemia and osteomyelitis	Staphylococcus	Resection left 4th, 5th and 6th cartilages	Recovered	
37	Tizon and Leroy[25]	M	12	Pneumonia (left)	Pneumococcus	"Method of Prof. Marquis"	Died, 1 day after operation	
38	Finney and Morgan[26]	M	15	? Pneumonia	Pneumococcus	Resection left 4th, 5th and 6th cartilages	Died, 19 days after operation	Developed empyema (right) 8 days after operation; autopsy showed anterior mediastinal abscess.

with chemotherapy; there is even less reason to expect suppurative pericarditis, similarly treated,

TABLE 3. *The Antecedent Disease in the Reported and Collected Cases of Suppurative Pericarditis.*

ANTECEDENT DISEASE	NO. OF CASES	RECOVERIES	DEATHS
Pneumonia	13	8	5
Pneumonia, with empyema	7	5	2
Empyema	5	3	2
Osteomyelitis	1	0	1
Osteomyelitis and septicemia	2	2	
Septic arthritis	1	0	1
Suppurative arthritis and septicemia	1	0	1
Metastatic abscesses (hip region and foot joint)	1	0	1
Tuberculous pericarditis	2	0	2
Influenza	1	1	0
Postoperative pneumonectomy for bronchogenic carcinoma and empyema	1	0	1
(?) Primary pericarditis	1	0	1
Stab wound	1	1	0
Ulcerative colitis and pyemia	1	0	1
Totals	38	20	18

to be cured. Nor is there any contraindication, as in acute empyema, to early open drainage.

The contrast of results in unoperated cases with those treated by pericardiostomy demonstrates that the latter offer a 50 per cent recovery rate, on the average, in collected series of cases. This is borne out in the most recently reported collected series of 99 cases by Shipley and Winslow,[1] in which 49 patients recovered. In the series of 38

TABLE 4. *Surgical Approach in the Reported and Collected Cases.*

APPROACH	NO. OF CASES	RECOVERIES	DEATHS
Anterior	29	16	13
Intercostal	1	1	0
Sternotomy	1	0	1
One cartilage and part of sternum	1	0	1
Two cartilages and part of sternum	1	1	0
One cartilage	6	4	2
Two cartilages	16	8	8
Three cartilages	3	2	1
Posterior	1	1	0
Epigastric	3	2	1
Two-stage: epigastric and anterior	1	1	0
Not stated	4	0	4
Totals	38	20	18

cases considered here, there were 20, or 53 per cent, recoveries, although in the 10 reported cases only 4 patients survived. These results should be improved in the next few years with a wider use of chemotherapy in conjunction with pericardiostomy. It is generally agreed that nonoperative measures deserve no place in the treatment of suppurative pericarditis.

In considering the problem of pericardiostomy, one has a choice of several methods of approach.

Intercostal approach. This has been largely abandoned because of ineffectual drainage, inability to secure dependent drainage and the impossibility of exploring or visualizing the pericardial sac.

Trans-sternal approach. As early as 1648, Riolanus suggested trephining the sternum as a means of draining the pericardium. Winslow and Shipley[32] advocated this approach, but in a later communication[1] inclined to resection of the costal cartilages. The disadvantage of this approach is the limited opening through the unyielding sternum, even though a portion of sternum is rongeured away.

Approach by resection of one or more costal cartilages, usually on the left. This approach is the one most commonly employed, and usually includes the fifth and sixth cartilages—the third to eighth cartilages have been resected. The transversus thoracis muscle fibers are divided, and the left pleural leaf is wiped lateralward, exposing the pericardium, which is incised, and the cut edges are sutured to the fascia or skin. In this method, pocketing of pus may occur posterior to the heart, or the heart itself may tend to occlude the drainage opening; however, placing the patient in the prone position may improve drainage. Occasionally, the corresponding cartilages on the right are resected. This was done in one of our cases (Case 4).

Xiphocostal or Larrey's angle approach, with resection of the seventh cartilage. This was later modified by Ogle and Allingham[33] to make their epigastric approach, in which the peritoneum is stripped from the diaphragm, and the pericardium entered dependently. This method has not been widely used, and we have had no personal experience with it, but it is said to give excellent dependent drainage, although, with the patient lying on his back, it seems to be subject, and for much the same reason, to the difficulties encountered in drainage by resection of the cartilages.

Posterior approach. Loucks,[34] Moore,[8] Truesdale[3] and Andrus[35] have reported favorably on this method, which is designed to drain the pericardial sac in its most dependent portion when the patient is recumbent. This approach requires traversing the left pleural cavity, and if this is not obliterated by adhesions, as it was in the cases of the operators noted above, the additional load of empyema thoracis is imposed and must be dealt with. This need not be insuperable, but the impression persists that this approach may best be used as a secondary operation, if pleural symphysis is not present.

Table 4 shows the approaches used in the 38 reported and collected cases.

There is no unanimity of opinion regarding the use of drainage material once drainage has been

established surgically. All types of materials have been used without any preponderance of evidence in favor of one or another. It is undoubtedly important that the pericardial cavity be explored with the finger at the time of operation, and at any later time that pocketing may be suspected. In 3 of our patients (Cases 3, 4 and 5), it was necessary daily to pass the finger around the heart to break up completely the fibrinous adhesions that formed regularly for the first five postoperative days. The patient was not upset by this procedure and complained of pain only when the finger pressed the pericardium anteriorly against the deep surface of the sternum. Some authors have placed Dakin's tubing or another type of tubing posterior to the heart. This has seemed to us a dangerous practice, and this impression was substantiated by an accident that occurred during the convalescence of one of our patients (Case 3). Although we have used no drainage tubing in our 5 personal cases, in Case 3 we experimented with various types of irrigating and suction tubing placed at the wound edges and not within the pericardial sac. Owing to a misunderstanding, a piece of 8-mm. red rubber tubing was placed within the pericardium, so that it was in contact with the inferior aspect of the right ventricle, and allowed to remain in place for approximately twelve hours. This resulted in a deep, eroded groove in the substance of the ventricle in contact with the tube, with ensuing brisk hemorrhage from the vessels in the eroded myocardium. It was necessary to take the patient to the operating room, and to rotate the heart outward and to the left before two silk sutures could be placed to control the bleeding. Fortunately, convalescence was uneventful thereafter.

As a result of this experience, we are more firmly than ever of the opinion that drainage or irrigating tubes, placed and fixed within the pericardium, are a definite hazard.

We are completely in accord with those who have stressed the value of frequent irrigations. We have employed gravity flow from a height of not more than 50 cm. above the pericardium, directing the fluid to the posterior portion of the sac by means of a No. 14 urethral catheter inserted at the beginning and removed at the conclusion of each irrigation. We have used a liter of Dakin's solution every three or four hours while purulent drainage continues, and have noted no such reactions as were suggested by the experimental work of Beck.[36] It has been used with apparently beneficial results by others,[33] and we have observed clearing of the pericardial infection with its use, whereas infections in other organs have persisted and the patient has gone on to die from sepsis (Case 4).

Prognosis

After studying the documented cases with respect to age of patient, sex, type of approach and postoperative care of the wound, one can determine no apparent effect of these factors on the prognosis. Although it seems reasonable to assume that those patients operated on earliest stand in the most favorable prognostic light, there is no weight of evidence to bear this out either in these 38 cases, or in those previously recorded. Shipley[37] states that of the 7 patients who recovered in his series, all were operated on early. On the other hand, of the 5 fatal cases only 1 was a late case.

We believe that prognosis depends on, first, the condition of the patient relative to the antecedent disease — as we have stated above, if the pericarditis is but a part of a pyemia, the outcome is usually fatal — and, secondly, the virulence of the infecting organism and, as a corollary, the resistance of the host. All the common pathogens and many of the rarer ones have been reported as the causative agent of suppurative pericarditis. Table 5 shows the organisms mentioned in the 38 reported and collected cases. It appears from this that the pneumococcus, with 11 recoveries and 5 deaths, is the most benign of the invaders, since, of the remainder, there were 9 recoveries and 13 deaths. These assumptions are not borne out in the literature. Undoubtedly, with the newer chemotherapy one may expect an appreciable improvement in these figures.

Table 5. *The Etiologic Agents in the Reported and Collected Cases.*

Organism	No. of Cases	Recoveries	Deaths
Pneumococcus, Type 1	2	1	1
Pneumococcus, Type 2	2	2	0
Pneumococcus, Type 4	1	1	0
Pneumococcus, Type 5	1	1	0
Pneumococcus, Type 8	2	1	1
Pneumococcus, type not stated	8	5	3
Staphylococcus albus	2	0	2
Staphylococcus aureus	7	4	3
Staphylococcus aureus (hemolytic)	1	0	1
Streptococcus hemolyticus	1	1	0
Streptococcus nonhemolyticus	3	2	1
"Germ of nature of colon bacillus"	1	1	0
"Appearance of *B. pertussis*"	1	1	0
Not stated	6	0	6
Totals	38	20	18

Case Reports

Authors' Cases

Case 1. C. S., a 48-year-old man, was admitted to the Third Medical Service of the Boston City Hospital on December 27, 1936. He gave a history of pneumonia of

4 days' duration. Physical signs of consolidation of the left lower lobe were present and were confirmed by roentgenogram. On December 31, 1936, a pericardial friction rub was heard over the entire precordium. On this day, also, the patient left the hospital against advice, and was apparently comfortable for a week. However, 1 week before re-entry on January 18, 1937, he began to have cough, fever, chills, sweats and pain in the left chest.

On physical examination, the heart was enlarged to percussion. There was a pulse deficit, and the sounds were distant and muffled. Later, pulsus paradoxicus and engorgement of the neck veins were observed, and the pericardial effusion was confirmed by roentgenogram. Pericardicentesis was performed, and 440 cc. of thick, greenish-yellow pus was removed. The venous pressure prior to pericardicentesis was 260 mm. of water, afterward, 170 mm. The patient was afforded temporary relief, and the next day 520 cc. of pus was removed from the pericardium. On the following day, he was transferred to the Second Surgical Service for pericardiostomy, which was carried out under nitrous oxide and ether anesthesia. The left 4th and 5th cartilages were resected, and the pericardium was opened. About 700 cc. of thick, yellow pus was evacuated. The pericardium was sutured to the skin; no drainage material was used. Postoperatively, the pericardial sac was irrigated with warm saline solution.

The patient showed considerable improvement, but by the 7th postoperative day an empyema was noted in the left thorax, from which a hemolytic streptococcus was cultured. Thoracentesis was performed on 4 successive days; meanwhile, the condition grew progressively worse, bronchopneumonia developed, and 10 days after operation the patient died.

Case 2. V. T., a 13-year-old girl, was admitted to the Second Medical Service of the Boston City Hospital on December 14, 1938. The patient had been well until 3 days prior to admission, when she complained of severe pain in the lumbar region and frequent urination. At the time of entry, she was febrile, flushed and dyspneic.

Roentgenograms demonstrated right-lower-lobe consolidation. Throat cultures showed a Type 6 pneumococcus, in addition to *Staphylococcus aureus*, and 200,000 units of Type 6 antipneumococcus serum were administered. In addition, the patient received sulfanilamide and, later, sulfapyridine. On December 18, two blood cultures, which had been taken previously, yielded *Staph. aureus*. In view of this, together with the failure to respond to serum therapy, it was thought that the patient had *Staph. aureus* pneumonia and septicemia. She was given 200,000 units of Lederle's antistaphylococcus serum, and there was transitory improvement. On the 8th hospital day, a pericardial friction rub developed, and extension of the pneumonic process was noted. Three days later, the area of cardiac dullness increased, and a pericardial tap yielded 4 cc. of serosanguineous material. Culture showed *Staph. aureus*. The patient was transferred to the Second Surgical Service, and pericardiostomy was performed on December 26. The 4th and 5th left costal cartilages were resected, and the pericardium opened. A small amount of pus and considerable fibrin were irrigated from the pericardial cavity. The pericardium was sutured to the skin, and no drainage material was inserted into the pericardial cavity. The pericardium was irrigated with warm saline solution.

Postoperatively, the condition continued to be grave; widespread bronchopneumonia with considerable pulmonary edema developed, and the patient died 42 hours after operation.

Case 3. W. B., a 33-year-old Negro laborer, was admitted to the Third Surgical Service of the Boston City Hospital shortly after having received multiple stab wounds of the chest on January 21, 1939.

On admission, he was in mild shock, and three stab wounds were noted, one over the midsternum, one in the region of the left 10th costochondral region, and one 2.5 cm. directly below the left nipple in the 5th left intercostal space from which there was moderate bleeding. Shock was relieved by heat, position, fluids and Coramine. The patient's progress was entirely uneventful until the 3rd hospital day, when he developed acute intestinal obstruction, for which laparotomy was performed under spinal anesthesia. The obstruction was found to be due to volvulus of the proximal ileum, and was relieved. The left leaf of the diaphragm was explored for evidence of injury, but none was found. There were no subsequent abdominal symptoms.

On the 8th day following entry, the wound beneath the left nipple, which had on occasion drained a small amount of bloody material, began to drain pus, and the patient became febrile. Cardiac enlargement, noted clinically, was later confirmed by roentgenograms. On February 11, examination showed — in addition to a chest wound draining pus — distended neck veins, paradoxical pulse, cardiac enlargement and a venous pressure of 170 mm. of water. The patient was transferred to the Second Surgical Service, and on February 12, under cyclopropane anesthesia, pericardiostomy was carried out. The left 5th and 6th cartilages were resected, the pericardium was opened, and approximately 1000 cc. of foul pus was aspirated. *Staph. aureus* was obtained on culture. The cavity was irrigated with physiologic saline solution, and the edges of the pericardium were sutured to the skin. No drainage material was placed in the pericardium. The patient showed immediate improvement.

The day following operation, an irrigating catheter was placed midway in the right border of the wound, but was not allowed to pass within the pericardium; the overflow was taken care of by another tube placed at the wound edge and connected to continuous suction. This system functioned well and kept the pericardial cavity bathed in fresh solution, at first, saline and, later, Dakin's solution. On February 23, the position of the catheter was changed to the lower angle of the wound and, inadvertently, allowed to remain in contact with the inferior aspect of the right ventricle for about 12 hours. The constant action of the heart against the catheter eroded the myocardium of the right ventricle. Two days later brisk hemorrhage from a myocardial vessel in the substance of the right ventricle occurred. It was discovered immediately, and two fine silk sutures were placed so as to occlude the bleeding vessel. Following this, the patient's recovery was steady, and by May 10 the pericardial wound was completely closed. However, there remained a short draining sinus down to the left 6th cartilage near the costochondral junction. Since the pericardium was well covered by skin, this sinus was excised. It filled in completely after several weeks.

Shortly before discharge, on June 26, a roentgenogram showed the size of the patient's heart to be normal. An electrocardiogram obtained in September, 1939, was within normal limits. The patient has been followed for 20 months since operation. He is well and working as a coppersmith. There is no evidence of adhesive pericarditis or impaired cardiac reserve on normal activity.

Case 4. H. S., a 44-year-old woman, had had ulcerative colitis for 10 months prior to her admission to the Boston City Hospital on June 25, 1940. There had been

a weight loss of 20 pounds. She was treated medically until August 23, when an ileostomy was performed. Following this, she had a very unsatisfactory course, since she could not be kept in fluid balance and the albumin-globulin ratio was reversed, the total serum protein being 4.5 gm. per 100 cc.; the blood chlorides remained about 85 milliequiv. per liter.

On October 2, a pericardial friction rub with pericardial effusion was noted, and at the same time there was evidence of sepsis in the right eye. Pericardicentesis was performed, and seropurulent fluid was obtained in which staphylococci were seen on direct smear. Pericardiostomy was performed on October 3, at which time her condition was poor. The 5th and 6th left costal cartilages were resected, and the pericardium was opened. Between 500 and 700 cc. of thin, yellow pus, which showed *Staph. aureus* and *Staph. albus* on culture, was evacuated.

The patient improved somewhat following this, but she continued to have frequent bowel movements. Ulcerations perforated the right cornea, and pus drained from the eyeball. She also developed a rectovaginal fistula. There was a small bloody effusion in the left pleura from which *Staph. aureus* was cultured, but which was well localized by aspirations. It was necessary daily, for the first 5 postoperative days, to break up, with the finger, fibrinous adhesions that formed between the heart and pericardium. The patient failed and died on October 11.

Autopsy revealed no infection of the pericardium. There were a localized collection of blood clots in the left costovertebral pleural gutter, panophthalmitis of the right eye and generalized peritonitis, the result of the perforation of one of the ulcerations of the ileum.

Case 5. J. P., a 15-year-old boy, was admitted to the Fourth Medical Service of the Boston City Hospital on October 15, 1940. He was desperately ill with a left-upper-lobe pneumonia of 2 days' duration, as demonstrated by physical signs and roentgenograms. Sulfathiazole therapy was begun, and a Type 5 pneumococcus was identified in the sputum. The blood culture taken on admission was also positive for Type 5 pneumococcus. There was definite improvement, but the temperature did not return to normal after 3 days.

On October 19, a pericardial friction rub was heard, and a roentgenogram showed definite pericardial effusion and also fluid in the left pleural cavity. There was marked pulsus paradoxicus. Pericardicentesis yielded no fluid, but the picture was so characteristic and the patient's condition so precarious that exploration of the pericardium was believed to be indicated, and he was transferred to the Second Surgical Service. Under local anesthesia, the 5th and 6th left costal cartilages were resected. Before the pericardium was opened, a needle was inserted and turbid straw-colored fluid obtained, which on direct smear showed numerous polymorphonuclear leukocytes but only questionable cocci. The left pleural lappet was fused and densely adherent to the pericardium, so that it was necessary to enter the pericardium by traversing the fused pleural layers. In so doing, no air was observed to enter the pleura, but a few cubic centimeters of fluid escaped. The pericardium, which was grossly thickened, was opened widely in the usual manner, and about 300 cc. of turbid fluid aspirated.

During the ensuing 2 weeks, many repeated cultures from the pericardium yielded Type 5 pneumococcus, although the cultures taken at operation were sterile. For the first 7 days, it was necessary daily to pass the finger around the heart to break up the fibrinous adhesions that had formed. On October 31, after several thoracenteses, a portion of the left 8th rib was resected, and the Type 5 pneumococcus empyema drained. Following this, convalescence was uneventful. The pericardiostomy wound healed in 40 days. The electrocardiogram was within normal limits, the empyema cavity obliterated, and the patient was discharged from the hospital on December 14.

Additional Boston City Hospital Cases*

Case 6. W. P. B., a 30-year-old physician, was admitted to the First Medical Service of the Boston City Hospital on January 18, 1913. He had had pneumonia 18 months previously, and at the time of admission was prostrated, dyspneic and cyanotic. On the following day, signs of consolidation in the right upper lobe were noted. After 3 days of high fever and a rather toxic course, the temperature dropped sharply to normal and the lungs cleared.

On the 5th day after entry, a pericardial friction rub was audible, and the patient was again febrile. Three days later, signs of pericardial effusion were present, and by January 31, 1913, the effusion had increased, the pulse became irregular and paradoxical, and the patient continued to be febrile. On February 18, aspiration of the pericardium yielded thick, yellow pus, from which pneumococci were cultured. On the same day, he was transferred to the Third Surgical Service, and pericardiostomy was performed under ether anesthesia by the late Dr. E. H. Nichols. The pericardium was entered after resection of segments of the right 4th and 5th costal cartilages, and 300 cc. of thick, yellow pus evacuated. A rubber-tissue cigarette drain was inserted into the pericardial cavity.

The patient was markedly improved following the operation, and his temperature at once became normal. On March 26, 1913, a portion of necrotic rib was removed, following which the wound promptly healed.

The patient at the present time (December, 1940) is in good health and actively engaged in practice as a dermatologist. There are no cardiac symptoms.

Case 7. S. E., a 17-year-old boy, was admitted to the Second Medical Service of the Boston City Hospital on November 27, 1917. Two weeks before entry, he began to have substernal and generalized pain throughout the chest, dyspnea, orthopnea and, later, chills, fever, sweating and anorexia.

On admission, the temperature was 102°F.; the patient was acutely ill, with a flushed face, but there was no dyspnea or cyanosis. At the base of the right chest posteriorly there was dullness, the breath sounds were diminished, and a few rales were heard. The liver was not enlarged, and there was no edema. The left border of the heart was 2 cm. beyond the mid-clavicular line in the 5th interspace, and the sounds were distant. The patient continued to be quite ill, and dyspnea and cyanosis developed.

Examination on November 30 revealed fluid at both bases, further increase in the area of cardiac dullness, and distant and muffled sounds; the liver was enlarged, and there was peripheral edema. On December 3, there was roentgenographic evidence of pericardial effusion. The patient continued to run a septic course and developed an irregular paradoxical pulse, the cardiac shadow increased in size, the cyanosis, dyspnea and peripheral edema increased, and ascites developed. On December 30, pericardicentesis was done, and 550 cc. of thin, greenish pus was obtained. The next day, pericardiostomy was per-

*We are indebted to Drs. H. A. Bouvé, E. D. Churchill and H. B. Loder for permission to report the cases of the patients on whom they operated.

formed under ether anesthesia by the late Dr. E. H. Nichols. A portion of the sternum and the 5th left cartilage were resected, and the pericardium opened. A rubber wick was inserted for drainage, and the wound was closed about the drain.

The patient was much improved by operation, and the pericardial wound drained profusely. He did well for 8 days, and there was a decrease in the amount of drainage. The wound became a small sinus, and completely closed on the 15th postoperative day. The patient then became irrational and edematous, developed bronchopneumonia, and died on the 28th postoperative day.

Case 8. B. C., a 14-year-old girl, was admitted to the Third Medical Service of the Boston City Hospital on August 15, 1921. Two weeks before entry, she had noted swelling, heat and tenderness of the distal phalanx of the right index finger, which discharged pus spontaneously. The patient was apparently well until 8 days before entry, when the right knee joint became painful, red and swollen. On admission, she appeared quite toxic. There were redness, swelling and tenderness in the distal phalanx of the right index finger, heat and swelling over the anterior portion of the right knee and marked tenderness on motion of the knee. The day following admission, under gas oxygen anesthesia, incision and drainage of the abscessed right index finger and right prepatella bursa were carried out. Cultures of the pus from each yielded *Staph. aureus.*

The patient continued to run a septic course, and on September 22, enlargement of the cardiac area was noted, and was confirmed by roentgenogram. The sounds were muffled, the pulse paradoxical, and there were compression signs in the left lung. Cyanosis, dyspnea, precordial pain, abdominal distention and free peritoneal fluid were present. The same day, pericardicentesis yielded 360 cc. of thick, grayish-green pus. The patient was markedly relieved following this, and on the next day, under ether anesthesia, pericardiostomy was performed by Dr. H. B. Loder. Segments of the left 5th and 6th costal cartilages were resected, and the pericardium was opened. A rubber tube was inserted into the pericardium.

Postoperatively, the patient failed to improve, continued a febrile course with increasing ascites, and died 23 days after operation.

Case 9. M. S., an 18-year-old student, was admitted to the Fourth Medical Service of the Boston City Hospital on December 5, 1930. One week before entry, he began to feel sharp pains in the ensiform region, made worse on breathing and accompanied by fever, headaches and anorexia. On the day before entry, the chest pain became worse, and there were dyspnea and a nonproductive cough.

On entry, the chest findings were consistent with a moderate bilateral effusion, and the heart was enlarged. Thoracentesis was done, and 100 cc. of slightly cloudy, yellow fluid was removed from the left chest. Cultures showed no growth. A febrile course continued, and on the 6th day, 450 cc. of similar fluid was removed from the left chest. On the 12th day, it was noted that the area of cardiac dullness was increased, and the pulse was paradoxical. At no time was a pericardial friction rub heard. At this time, there were definite signs of fluid in the right thorax, and thoracentesis yielded 1250 cc. of slightly cloudy, yellow fluid from which no organisms could be cultured. On the 19th hospital day, fluid was again removed from the right thorax, and at this time the area of cardiac dullness was very definitely increased. This was confirmed by a roentgenogram, which revealed enlargement both to the right and to the left. On December 24, pericardicentesis yielded 20 cc. of yellowish purulent fluid. Smear showed no organisms, and culture revealed no growth. By that time, the patient was very much worse; dyspnea, orthopnea and cyanosis were increasing. Two days later, a pericardiostomy was performed by Dr. E. D. Churchill, under local anesthesia. Segments of the left 6th and 7th costal cartilages were resected, and a very thick, fibrous pericardium exposed and opened. A large quantity of pus was evacuated. Cultures from this yielded *Staph. aureus.* A tube was inserted just within the pericardium.

The patient experienced immediate relief, and his postoperative course was uneventful. He was discharged from the hospital on the 48th postoperative day, symptom free and with the wound healed.

Case 10. F. L., a 12-year-old boy, was admitted to the Pediatric Service of the Boston City Hospital on June 8, 1930. He gave a history of malaise, fever and chills.

The entire right chest showed flatness, decreased breath sounds and decreased vocal and tactile fremitus. A roentgenogram revealed uniform density of the entire right chest, with displacement of the heart and trachea to the left. On June 10, the empyema was drained by the intercostal closed method, and 1400 cc. of pus was obtained from which a Type 1 pneumococcus was cultured. Following this procedure, there was some improvement, although the patient continued to be febrile.

On June 19, physical examination showed cardiac dullness extending into the left midaxillary region; the heart sounds were faint and indistinct. A diagnosis of pericarditis was made, and pericardicentesis on June 19 yielded pus containing Type 1 pneumococcus. On the same day, pericardiostomy was performed by Dr. H. A. Bouvé. Under local anesthesia, "a cartilage was resected and a catheter inserted into the pericardium through a trocar."

The patient failed to improve postoperatively, and died on the 15th day following operation.

Summary and Conclusions

Ten cases of suppurative pericarditis treated by pericardiostomy and hitherto unreported are presented.

Twenty-eight additional cases have been collected from the literature from January 1, 1934 to January 1, 1940, which, with the above 10 cases, brings the total of recorded cases to 265.

Suppurative pericarditis is not a rare disease, as is demonstrated by the autopsy figures.

The diagnosis is frequently missed, because the condition is obscured by concomitant thoracic disease.

In untreated cases, the mortality approaches 100 per cent.

With pericardiostomy, the treatment of choice, the mortality is 50 per cent.

With chemotherapy as a supplement to pericardiostomy, there should be a further reduction of mortality.

Pericardiostomy should be undertaken early to achieve optimal surgical conditions and probable further lowering of mortality. Such a policy may involve operating on a certain number of patients in whom the diagnosis cannot be proved by peri-

cardicentesis, and even in a few in whom organisms cannot be found in the fluid obtained by early pericardicentesis.

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DYSPHAGIA AND NUTRITIONAL DEFICIENCY*

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THE most significant causes of dysphagia are carcinoma and other malignant tumors of the esophagus, cardiospasm or achalasia of the esophagus, benign esophageal stenosis (or stricture), hypochromic anemia, with the Plummer–Vinson syndrome, and paralysis of the ninth or tenth cranial nerves. Dysphagia very rarely occurs in association with a dilated left auricle, pericarditis, a saccular aneurysm, a dissecting aneurysm or an anomalous aortic arch.[1] A certain degree of dysphagia may also be present in gastric diaphragmatic hernias, especially in those cases in which there is a congenitally short esophagus or in which the lower end of the esophagus is kinked as a result of hernia.[2]

Since cancer of the esophagus is one of the most inexorable forms of neoplastic disease and since most of the other conditions causing dysphagia are amenable to appropriate therapy, it is imperative to establish the etiology of a given case so that the proper treatment may be promptly instituted.

Recently, several cases have come to our attention that at first were thought to be cancer of the esophagus but on further study were found to have dysphagia apparently due to polyneuritis secondary to nutritional deficiency. An adequate diet, with vitamin supplements administered by mouth, by stomach tube or by gastrostomy tube, as the situation necessitated, resulted in amelioration of the polyneuritis and disappearance of the difficulty in swallowing.

This aspect of deficiency disease has rarely been stressed in this country, but seems to be sufficiently important to warrant serious consideration.

In 1924, Vinson,[3] reporting 415 cases of "cardiospasm," said that the etiology is unknown but "among the causes may be primary spasm, primary atony of the musculature of the esophagus, *irritative lesions of the vagus nerves* [italics ours], esophagitis, fissures of the cardia, kinking of the esophagus at the hiatus esophagi and extrinsic pressure from the liver."

In 1938, Carr and Vinson[4] considered cardiospasm to be due to either vagal hypoactivity or a sympathetic hyperactivity, or both, but they admitted that the underlying cause of the autonomic imbalance is unknown.

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Knight,[5] studying the physiology of the esophagus in cats, concluded:

> (1) The esophagus receives a sympathetic innervation. (2) There is a true intrinsic sphincter at the cardia. (3) Vagal stimulation causes tetanic contraction of the upper one third of the esophagus, which is composed of striated muscle. This contraction is augmented by sympathetic stimulation. (4) Vagal stimulation causes increased tonus and motility of the lower one third of the esophagus, which is composed of smooth muscle. The tonus and motility are inhibited by sympathetic stimulation. (5) Bilateral vagal section reproduces the appearances of achalasia of the cardia. Simultaneous removal of the sympathetic fibres prevents the onset of this obstruction. When the obstruction develops it can be relieved by section of the sympathetic supply to the sphincter. (6) The sympathetic supply of the cardiac sphincter is accessible as it passed to the sphincter along the course of the coeliac axis and left gastric arteries.

Pollitzer[6] reported a case of marked esophageal dilatation in a sixty-seven-year-old woman who had had dysphagia for thirty years. Autopsy showed compression of the vagus nerves by enlarged mediastinal lymph nodes. He believed that pressure on the vagus nerves had caused dilatation of the esophagus.

Various authors[7–12] have reported pathologic findings in Auerbach's plexus in post-mortem studies of cases with achalasia of the esophagus. Constant findings were degeneration of this nerve plexus and hypertrophy of the circular muscular layer of the esophagus. Occasionally, in addition, small collections of lymphocytes and plasma cells, which suggested a certain amount of chronic inflammation, were found scattered throughout the wall of the esophagus.

Lendrum[13] has pointed out that the loss of ganglion cells in Auerbach's plexus is as great in the undistended portion of the lower esophagus as in the distended portion. This fact suggests that the pressure associated with the distention is not the cause of the degenerative changes in the nerve plexus.

Looser[14] reported a case of cardiospasm with enormous dilatation of the esophagus in a twenty-one-year-old woman whose symptoms dated back to diphtheria at the age of twelve. He mentions no paralyses in this case, and he effected a cure by forcible dilatation of the lower end of the esophagus after performing a gastrostomy.

In view of the present-day knowledge of the significance of nutritional deficiency in the etiology of many types of polyneuritis, it is of great interest that Vinson,[15] reporting 186 cases of cicatricial (benign) stricture of the esophagus in 1927, noted the following etiologic factors: 18 cases had no obvious cause for stricture, 14 followed typhoid fever, 13 followed severe vomiting of pregnancy, and the remaining 141 were accounted for by various caustics, congenital defects and infectious diseases. In other words, 27 cases out of 186 (14 per cent) developed following typhoid fever or pernicious vomiting of pregnancy, conditions that are now known to be frequently associated with manifestations of deficiency disease. Vinson does not, however, include sufficient clinical information to enable one to estimate the incidence of recognizable deficiency-disease syndromes among his patients.

Turner,[16] reporting on 19 cases of nonmalignant esophageal stenosis, found that 5 were due to ingestion of corrosives, 5 were congenital, 4 were due to acute inflammation, and in 2 the cause was undetermined. He also said that simple strictures have followed scarlet fever, typhoid fever and the vomiting of pregnancy, but offered no explanation of this unusual sequence of events.

Stinson,[17] in 1938, reported 4 cases treated and cured with thiamin chloride. He came to the conclusion that cardiospasm is a manifestation of vitamin B_1 deficiency, that vitamin B_1 given intramuscularly relieves certain types of cardiospasm, and that the continued administration of this vitamin prevents the recurrence of attacks. Unfortunately, his paper includes no histories of dietary deficiency, nor does he include descriptions of physical signs of the better-known syndromes now accepted as being due to vitamin deficiency. From the evidence given, it is hardly justifiable to consider that he has proved the etiologic relation of vitamin B_1 deficiency to cardiospasm.

Meiklejohn[18] has recently cast some doubt on the experimental evidence usually quoted as proving that thiamin (vitamin B_1) is actually the antineuritic vitamin, but he concedes the relation of dietary deficiency to certain neuritides.

Fenwick,[19] in 1898, published a paper discussing spasmodic stricture of the cardiac orifice of the stomach. He noted: "Habitual drunkards are very liable to the complaint especially after indulgence in an unusually deep debauch. In many of these cases a bougie is only arrested in the immediate neighborhood of the stomach, but in others the obstruction is encountered just below the pharynx." He mentions one case in a youth with acute phthisis, and also says that dysphagia sometimes appears following an acute febrile illness. Of some historical interest is his comment that a mild form of the complaint is frequently encountered in emotional and anemic women between the ages of eighteen and thirty, in some cases making its appearance coincident with the establishment of the menses. Perhaps he refers here to two

conditions that would now be diagnosed as globus hystericus and idiopathic hypochromic anemia with the Plummer–Vinson syndrome.

Jankelson[20] reported 6 cases of dysphagia that were relieved by the administration of vitamin B. One of his patients had severe polyneuritis, another had paresthesias and weakness of the arms, and a third had pain in the calf muscles and marked general weakness. The others had no demonstrable neuritis. Four patients had no x-ray evidence of disease in the esophagus or stomach, one had a small diverticulum in the middle third of the esophagus, and the sixth case had no x-ray studies reported.

Manson-Bahr,[21] in discussing the nerve lesions of beriberi, says that the cranial nerves above the seventh are rarely involved, but in some cases the laryngeal muscles are paralyzed. In the infantile form of beriberi, laryngeal paralysis is believed to be relatively common, and dysphagia and aphonia may be present.

Vedder,[22] discussing the pathology of beriberi, states that degenerative changes are found in the posterior columns as well as in the anterior and posterior nerve roots and ganglion cells. He adds, "It is especially to be noted that degeneration of the sympathetic system has been demonstrated, as in branches of the cardiac plexus, the splanchnic nerves and branches of the solar and renal plexus."

Laryngeal symptoms have been reported in beriberi by Fernando,[23] and paralysis of both recurrent laryngeal nerves in beriberi by Miura.[24]

Etzel[25] and Netto[26] believe that achalasia of the pylorus in the adult is identical in etiology and pathologic physiology with megaesophagus (achalasia of the esophagus), hypertrophic pyloric stenosis in the newborn, and megacolon, these conditions being due to vitamin B_1 deficiency. The pathology in all is the same and consists in marked diminution of elements of Auerbach's plexus.

Brodie,[27] in some experimental work on rats on the etiology of congenital pyloric obstruction, was able to produce this condition in 10 out of 23 young rats (four litters) by keeping the mother's diet minimal in antineuritic vitamin (B complex). The vagus nerves of the affected rats showed myelin degeneration.

The preceding observations suggest that dietary deficiency may be a cause of dysphagia, although the mechanisms involved may vary.

Iron-deficiency anemia associated with dysphagia causing the Plummer–Vinson syndrome has been so frequently reported that no further mention of it will be made here, even though the mechanism by which the dysphagia is brought about is anything but clear.

The following 3 cases are reported as examples of dysphagia secondary to nutritional deficiency.

Case Reports

Case 1 (formerly reported by one of us [D. M.[28]]). R. W. (P. 7094), a 55-year-old laborer, was transferred from a large teaching hospital with a diagnosis of carcinoma of the esophagus. Two and a half months before admission, he developed a rapidly progressive dysphagia and became barely able to swallow liquids. During the same period, he lost weight and developed a generalized weakness of all the muscles, particularly of the hands and feet, associated with tingling and numbness during the last 4 weeks. He had been studied in the neurologic division of the other hospital, and diagnoses of cancer of the esophagus (on the basis of fluoroscopic examination), toxic paralysis of the 10th cranial nerves, and toxic myelitis had been made. The diagnosis of 10th-nerve paralysis was based on hoarseness, regurgitation of fluids through the nose and weakness of the left side of the palate.

The past history included exposure to tuberculosis 5 years before admission. The patient had worked for many years handling bags of lime in a very dusty atmosphere. For 5 years, he had had a "cigarette cough." He had been a heavy drinker of alcoholic beverages since the age of 14.

On examination, the positive findings were dullness, increased tactile fremitus and fine crackling rales over the right chest posteriorly. There was generalized atony and wasting of the large calf and thigh muscles and intrinsic muscles of the hands. The deep tendon reflexes were absent, the abdominal and cremasteric reflexes barely perceptible. There was slight diminution of vibration perception over both legs below the knees, and moderate tenderness to pressure along the deep nerve trunks in the thighs, calves and arms. There was a definite tendency to bilateral foot drop.

The significant laboratory findings were a red-cell count of 2,730,000 with a hemoglobin of 56 per cent (Sahli), and a white-cell count of 11,500 with a normal differential.

X-ray examination of the chest showed dullness of the upper half of the left lung and upper third of the right lung, which was interpreted as being due to tuberculosis or to metastatic carcinoma. Fluoroscopy of the esophagus showed a deformity of the first portion, without marked obstruction, and was thought to represent a large tumor projecting into the lumen of the pharynx. Studies repeated 2 weeks later showed no change. Laryngoscopy under general anesthesia showed a markedly thickened fold of mucous membrane that had the appearance of a web just behind the larynx. No ulceration was seen, and a No. 18 bougie passed readily through the esophagus.

Because of the evidence of polyneuritis and a history of alcoholism, the patient was started on a high-vitamin diet, with liver extract intramuscularly as a source of vitamin B. Although bedridden for 3 weeks after admission, he improved steadily so that he was able to stand and walk a little by the end of the 4th week. Then, because he was much stronger, esophagoscopy was performed but no disease was found; this was repeated and again was negative. It was also noted at this time that the patient could swallow even solid foods without difficulty.

Six weeks after admission, another x-ray film of the chest was taken. The entire right lung was clear, only a few increased markings persisting at the left apex. The blood showed only slight improvement, in spite of liver and iron therapy. The red-cell count rose to slightly over 3,000,000, and the hemoglobin to 65 per cent.

During the 8th week, the patient developed an acute respiratory infection, with signs of congestion in both lungs. He became unco-operative, drowsy and confused. Neurologic examination and lumbar puncture were negative except for absent tendon reflexes, which had never returned. The course was then rapidly downhill. The patient developed what was thought to be a bronchopneumonia and died 12 weeks after admission.

A post-mortem examination revealed multiple abscesses in both lungs, terminal bronchopneumonia, a normal esophagus and a cystic tumor of the right frontal lobe, which on microscopic examination proved to be a fibrillary astrocytoma.

It is interesting that normal esophageal function as demonstrated by x-ray and esophagoscopy returned while the patient was receiving large amounts of vitamin B complex, although he was suffering from a brain tumor and multiple lung abscesses that eventually caused his death.

Case 2. E. A. (P. 16023), a 66-year-old man, was admitted because of progressive dysphagia of 3 weeks' duration. He had been an inmate of a state institution for 4½ years, with a diagnosis of manic-depressive psychosis. The throat trouble began as a "sore throat," but difficulty in swallowing soon progressed to the point where even fluids were regurgitated through the nose. The patient had lost 20 pounds in the previous 2 months.

Physical examination was essentially negative except for dental caries and external hemorrhoids. Nutrition appeared to be excellent.

Because the patient was totally unable to swallow, a gastrostomy was performed. Postoperatively, he did extremely well and gained weight rapidly. However, about 1 month after operation he complained of tingling of his hands and feet, his gait was unsteady, and he lost all position sense in his legs. His tongue was noted to be atrophic. Esophagoscopy revealed only a slight constriction about 25 cm. from the teeth, with no evidence of cancer, although a barium swallow was promptly regurgitated and spilled into the trachea.

On gastrostomy feedings with added liver, sensation in the extremities returned, the gait became normal, and the patient was able to swallow liquids without difficulty. He was discharged back to the state institution 2 months after operation, with a diagnosis of esophageal spasm and stricture. He returned to the Out-Patient Department 5 weeks later for a check-up examination, at which time he was able to swallow solid foods without any trouble; the tongue was normal, but the knee jerks and biceps reflexes were absent. There was also a fine scaling desquamation of the extensor surface of the shins and forearms. A diagnosis of vitamin B deficiency, with polyneuritis and paralysis of the 10th nerve, was made, and brewer's yeast (four 7½-gr. tablets after each meal and at bedtime) was prescribed.

The patient was seen again 1 month later, at which time he had gained 11 pounds and was able to eat anything without difficulty; there was marked improvement of the paresthesia of his hands and feet. As he described it, he was able to wind his watch and know what he was doing. All evidence of dermatitis had disappeared, and the deep tendon reflexes, although sluggish, were present. Esophagoscopy was repeated, and since no lesion was found, the gastrostomy was allowed to close.

The patient was last seen 7 months later, 11 months after the gastrostomy had been performed; he had gained another 4 pounds and had no symptoms or signs of any residual neuritis, and no symptoms referable to his esophagus.

In retrospect, it seems likely that the extensive neuritis and loss of 20 pounds of weight had been caused by the patient's failure to eat adequately during a depressed phase of his manic-depressive psychosis.

Case 3. J. W. (P. 10181), a 48-year-old man, was first seen in December, 1935, because of hoarseness of 4 months' duration, gnawing epigastric distress, increasing weakness and tingling, and numbness and coldness of the arms and legs. He had been able to take only milk and coffee and had had marked anorexia for the same length of time. He admitted excessive indulgence in alcohol for many years. Thirty-three years prior to admission, he had had "an operation on his stomach," and 28 years later he had had "an operation for ulcer of the stomach."

The striking findings on examination were marked hoarseness and inability to stand or walk without assistance. Neurologic examination showed hyperactive but equal knee and ankle jerks, and marked muscular weakness; efforts made to stand up resulted in clonic contractions of the leg muscles.

Laryngoscopy showed the vocal cords to be a little sluggish in motion, and there was a small tumor at the base of the epiglottis. A biopsy of this tumor showed only chronic inflammation.

The patient was placed on a high-vitamin diet, with marked improvement of the neuritis. His appetite was excellent, he was able to swallow without difficulty, his hoarseness disappeared, and he was able to walk without support, though unsteadily, when he was discharged 1 month after admission. The final diagnosis was polyneuritis due to nutritional deficiency secondary to chronic alcoholism.

The patient was next seen again about 1½ years later, when he came to the Out-Patient Department because of pain in the hypopharynx, difficulty in swallowing and hoarseness. Laryngoscopy showed marked congestion of the entire hypopharynx but no cancer. In December, 1939, after an interval of almost 2½ years, the patient was admitted from a state prison because of constant burning pain in the epigastrium, pain in the left lower quadrant of the abdomen and burning pain in the suprasternal notch. For the previous 2 years, he had been unable to eat anything except liquids and soft foods. For 14 months, he was at a veterans' hospital, where in June, 1939, because of inability to swallow, a gastrostomy had been performed. Although the gastrostomy had closed over, the patient had apparently gained 33 pounds in 6 months and could swallow better than before the operation. For the previous 12 months, he had been receiving hypodermic medication at 4-hour intervals because of abdominal pain. He had been in bed practically the entire time because of weakness. He could not lie flat in bed, however, because his throat filled up with mucus when he did.

Physical examination was surprisingly negative, considering the dramatic history. The liver edge was palpable 3 fingerbreadths below the costal margin. There were coarse tremors of the tongue and of all extremities. The knee and ankle jerks were equal and hyperactive. Gastrointestinal studies, including a barium meal and enema, were negative for any evidence of neoplastic disease. The blood Hinton reaction was negative. On a

high-vitamin diet containing brewer's yeast (six 5-gr. tablets after each meal and at bedtime), the patient improved rapidly, and was soon able to sit up and to walk when supported. He gained another 6 pounds in the 3 weeks at the hospital, and was discharged with no difficulty in swallowing and complete relief of the burning in the suprasternal notch, epigastric discomfort and left lower-quadrant pain. His strength was returning rapidly, but he still had marked muscular inco-ordination in his legs.

The discharge diagnosis was the same as that on the first admission to the hospital: polyneuritis due to nutritional deficiency secondary to chronic alcoholism. The dysphagia and aphonia were apparently due to involvement of the 9th and 10th cranial nerves by the polyneuritis.

Conclusions

Dysphagia is occasionally a result of neuritis involving the ninth or tenth cranial nerves, which in turn is produced by nutritional deficiency, particularly of the vitamin B complex.

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LEFT-SIDED PAIN IN BILIARY-TRACT DISEASE*

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THE pain of biliary-tract disease is primarily epigastric or right-sided. Occasionally, however, there is a left-sided component in the pattern of pain reference. When this happens, either pancreatitis or an extension of the pericholecystic inflammation to the left side is generally a sufficient cause. However, pain only on the left side may occur in biliary-tract disease in the absence of an inflammatory process. In such cases, which are rare, the origin of the pain in the biliary tract may be overlooked because of the clinical habit of regarding the latter as possessing an exclusively right-sided innervation. On embryologic grounds, the common duct must be assumed to have a bilateral innervation. This provides a reasonable explanation for some cases of bilateral pain in noninflammatory disturbances of the biliary ducts and for the occasional case in which there is an exclusively left-sided reference. This is not a new observation, but two striking clinical illustrations are recorded herewith to emphasize it and thus to minimize the likelihood of a misinterpretation of the existing disease in similar cases.

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Case Reports

Case 1. A 45-year-old woman was admitted with a history of severe stabbing pain of 8 years' duration, starting in the epigastrium and radiating around the left costal margin and to the left scapula. During the more intense seizures, pain was also felt in the left shoulder and arm, spreading to the finger tips. The onset of attacks bore no relation to the ingestion of food, to the kind of food, to bowel, bladder or catamenial functions, to rest or to exercise. The pain, instead of immobilizing her, caused her to roll about in bed; it was not colicky, but rather constant, lasting from 30 minutes to 12 hours. It was not relieved by food or alkalies. There was no nausea or vomiting. Six years before entry, following an attack in which slight jaundice was noted, a cholecystectomy was performed in another hospital. The gall bladder contained numerous stones and gravel. The surgeon's note was as follows: "A moderately thickened and enlarged gall bladder was adherent to the whole length of the

duodenum. The common-duct and cystic-duct regions were explored and examined. The gall bladder was removed. The common duct was negative and was not drained. The pancreas was normal." Following operation, the patient had attacks of acute pain in the chest, which were diagnosed as angina or pseudo-angina by a consultant. When last seen by him 1 year after the operation, she was still having severe attacks of pain, "cutting off her breath," and the consultant was not certain what the diagnosis was.

The patient was admitted to this clinic 6 years after the cholecystectomy. She stated that 10 days after the cholecystectomy she had an attack of pain exactly like those preceding the operation. The attacks continued as frequently and as severely as ever, but during the previous few years there had been no radiation of pain to the left scapula and left upper extremity. The pain began just below the xiphoid process, radiated along the left costal margin and caused difficulty in breathing. Following the seizures, she felt a girdlelike, squeezing sensation, which slowly subsided. There was no nausea or vomiting and no other symptoms referable to the gastrointestinal system. No jaundice was noted at any time. Amyl nitrate occasionally relieved the pain; often it did not. Between attacks there was no intolerance of food, heartburn, flatulence or constipation; the appetite was normal. There had been an attack of pain almost daily for the 2 weeks preceding admission.

Physical examination on admission was not remarkable except for the scar of the incision in the right upper quadrant and another of an appendectomy done when the patient was 16.

The stools, blood and urine showed normal findings; the icteric index was 6, and the blood Hinton and Kahn reactions were negative. Gastric analysis gave normal findings; the spinal fluid and the sugar-tolerance curve were normal; and the electrocardiogram was negative. Radiologic study of the genitourinary tract, chest, spine, stomach and intestine gave findings within normal limits. Duodenal intubation yielded crystals of calcium bilirubinate and others suggestive of cholesterol. Accordingly, the patient was explored, with a preoperative diagnosis of choledocholithiasis. After old adhesions in the region of the gall-bladder bed had been freed, the common duct, which was slightly dilated and thickened, was opened and a single pea-sized gallstone found and removed from the lower end.

The patient recovered uneventfully and has been entirely symptom free, enjoying excellent health ever since, a postoperative interval of 2 years.

Comment. The exclusively left-sided radiation of pain, in the absence of jaundice, nausea, vomiting or other symptoms that might have suggested a diagnosis of choledocholithiasis, was so misleading that no physician suggested the possibility of residual stone in the biliary tract until after 6 years of continued disability.

Case 2. A 71-year-old woman entered the hospital because of pain, colicky in nature, radiating around both costal margins and to the interscapular area in the back. A week before entry, she experienced her first attack, in which the pain was in the right upper quadrant and radiated to the back between the scapulas. In the second attack, lasting 9 hours, which occurred on the day of admission, the pain was more pronounced on the left side, but it radiated to both sides, giving her the "feeling of a rope being tied around the waist." She found no relief in any position. There was some nausea, but no vomiting. There was no history of chills, fever or jaundice. The patient had been treated for 2 years for attacks of substernal distress, pain in the left shoulder and dyspnea on exertion. These were attributed to coronary heart disease.

On admission, physical examination revealed tenderness and spasm in the right upper quadrant of the abdomen. The electrocardiogram showed sinoauricular bradycardia (rate, 55), left-axis deviation, widened QRS complexes and probable intraventricular block. The urine contained a few red and white cells per high-power field, and the blood white-cell count was 8900. The icteric index was 15. A Graham test showed cholelithiasis.

For 6 days preceding operation, the patient had nightly seizures of bilateral girdle pain, of equal intensity on both sides. Cholecystectomy and common-duct exploration were then performed. A small gall bladder containing numerous stones was excised, and several stones were removed from a dilated and thickened common duct. T-tube common-duct drainage was established. There was no pancreatitis or evidence of an acute inflammatory process in the upper abdomen.

The convalescence was smooth until the 8th postoperative day, when clamping of the T-tube caused severe discomfort, which was quickly relieved by unclamping the tube. Two days later a cholangiogram showed a residual common-duct stone. In view of the patient's age and cardiac condition, further surgery was deferred. She was discharged with the T-tube in place.

When seen 7 weeks later, she looked well. She stated that about 100 cc. of bile came out of the tube in each 24 hours. She had been entirely free of all symptoms, including the precordial pain attributed preoperatively to the coronary heart disease, although she acknowledged that exertion, the usual preoperative cause of the pain, had been avoided. Before operation, however, there had been some spontaneous pain in the left chest, which had not recurred since. To establish a possible relation between the common-duct stone and the left-sided pain of which she complained in the immediate preoperative period, 30 cc. of physiologic saline solution was injected slowly into the T-tube. She complained immediately of pain along the left costal margin and pain in the back below the angle of the left scapula. This procedure was repeated using 45 cc. of saline injected rapidly, with the reproduction of the same pain in the same area. There was no pain reference whatever to the epigastrium or to the right side. The saline solution in both instances did not return to the through tube and therefore presumably passed by the stone and entered the duodenum. Incidentally, this procedure succeeded in forcing the stone into the duodenum, since a subsequent cholangiogram showed no stone. The tube was removed, and the patient has remained well to date.

Comment. This is a clinical experience demonstrating beyond doubt that distention of the common duct can cause exclusively left-sided pain. It behooves the physician not to dismiss the common duct from consideration in the differential diagnosis when the presenting symptom is pain in the left upper quadrant.

Summary and Conclusions

In two patients with stone in the common duct, pain was referred only to the left side in one and to both sides with equal intensity in the other. In the latter case, distention of the common duct postoperatively produced pain on the left side only.

Common-duct obstruction, therefore, may cause pain referred only to the left side and in the ab-

sence of other signs or symptoms suggesting disease in the biliary tract. It is not always necessary to assume the presence of pancreatitis or some other inflammatory process spreading to the left of the mid-line to explain left-sided pain in disease of the biliary system.

ANTHRAX*

Report of a Case Treated With Antiserum and Sulfathiazole

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ALTHOUGH satisfactory progress has been made in the treatment of anthrax, this disease continues to be a serious industrial hazard. The greatest number of cases occurs among tannery workers, but those among wool handlers and agricultural workers are not far behind. Although the fatalities are lessening in a few states,[1] the general mortality shows no tendency to decline, in spite of the fact that effective treatment is available.

Anthrax has been known since ancient times, mention being made in the Bible and by authors such as Homer, Seneca and Ovid. Although the descriptions reveal that even at that time the transmissibility of the disease to man by the fur and wool of diseased animals was known, it was not until 1823 that Barthelemy proved its contagious character in animals.[2] Pollender, in 1849, was the first to observe organisms in the blood of animals with anthrax.[3] In 1850, Davaine and Rambert demonstrated experimentally the relation of anthrax to the malignant pustule.[3, 4] This work was confirmed by Koch in 1876 and Pasteur in 1877.[3, 4] It was with the aid of the new methods devised by Koch that a firm foundation was laid for further investigation of the etiology and spread of anthrax.

Anthrax is a specific, virulent, infectious disease, caused by the *Bacillus anthracis* and occurring in man in three forms[2, 5–7]: the cutaneous, the pulmonary and the intestinal. The disease is communicable to man by contact, inhalation or ingestion of material infected with anthrax. The cutaneous form, also known as "external anthrax," is further subdivided into the malignant-pustule type and malignant anthrax edema. The intestinal and pulmonary types are also called "internal anthrax."

By far the commonest type is the cutaneous infection, usually occurring as the malignant pustule. The typical malignant pustule is a single lesion beginning with intense itching at the point of inoculation, or a burning sensation followed by the appearance of a small red papule, which within twelve or fifteen hours becomes vesicular, the vesicle showing a central depression containing brownish fluid and surrounded by a slight edematous areola. The vesicle then dries, leaving a dark-brown eschar surrounded by a number of minute silvery vesicles. The tissue about the site of inoculation becomes swollen, owing to anthracomucin, and consists of a doughy, indurated, pinkish, edematous mass. The surrounding lymph nodes are generally swollen, painful and covered by edematous skin. It should be noted that a characteristic feature of the pustule itself is the absence of pain in and about the lesion. Even when the lesion is extensive, the patient may complain of only a sense of weight and slight tenderness on pressure, or a feeling of tightness because of swelling. The surrounding vesicles then become filled with hemorrhagic fluid, and the edematous area is infiltrated with blood and sometimes undergoes gangrenous degeneration. If the infection becomes generalized, all the symptoms of collapse eventually set in, the patient becomes pulseless, and respiration ceases.

Internal anthrax occurs far less commonly, intestinal anthrax being the rarest form. The pulmonary type is brought about by the inhalation of dust impregnated with anthrax bacilli, and is usually seen in woolsorters; there are pain in the chest, cough, expectoration of bloody sputum, and cyanosis. The intestinal type is usually produced by the ingestion of spores, and the symptoms of the disease are referable to the pulmonary and intestinal tracts. In the intestinal form, there are abdominal pain, nausea and vomiting and diarrhea. The constitutional symptoms of internal anthrax are severer than those in the simple malignant-pustule form.

The diagnosis of anthrax can be made only after all the criteria have been considered. A history

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of exposure to infection, followed by a physical examination presenting the clinical picture of anthrax, does not relieve the physician from performing bacteriologic tests that confirm the diagnosis. One must make a direct examination of the pustular secretion in the malignant-pustule form, of the sputum in pulmonary anthrax and of the feces in intestinal anthrax. Furthermore, only a positive stained smear is decisive. If the direct examination is negative, a culture should be made or a guinea pig should be inoculated before making a diagnosis including or excluding anthrax.

When the diagnosis of malignant pustule has been made, the question of therapy arises. It has been shown by recent experience[8–10] that any form of operation or cauterization is to be avoided, for such procedures permit the entrance of infectious material into the general circulation because of the severance of blood vessels and lymph channels, thus precipitating septicemia. It is known that the pustule of cutaneous anthrax often heals completely if no local interference is offered, and Didiée[11] reports good results from the use of small doses of x-ray.

The accepted local treatment consists in thorough cleansing of the lesion with physiologic saline solution, absolute rest and immobilization of the affected part and the injection of antianthrax serum subcutaneously at four points fairly equidistant from the lesion, as advocated by Symmers[12] and by Regan.[13] These injections form an isolating barrier against anthrax bacilli, and should be repeated at intervals of four or six hours until the lesion is free of bacilli. The amount of serum to be used depends on the severity of the local inflammation. The lesion should then be covered by sterile gauze. Every cutaneous anthrax lesion is to be regarded as a potential generalized infection, and an intravenous injection of 150 to 200 cc. of antianthrax serum should be given, due care being taken that the patient is not sensitive to the serum. If the blood culture is found to be negative, no further intravenous injections are necessary. If one errs, it is thus on the side of safety, for valuable time is saved in the event of septicemia.

For the treatment of cutaneous anthrax with septicemia, in addition to local therapy, as described above, repeated intravenous injections of antianthrax serum are recommended. Favorable results have been reported from the use of relatively small doses of serum; however, as stated by Symmers[12]: ". . . it would appear that to depend upon the lesser quantities as a routine measure is sooner or later to court disaster — and in dealing with a disease so treacherous as anthrax, it is well to leave a wide margin of safety." A proved, efficient therapeutic method for treatment consists in the immediate administration of 200 cc. of serum, repeating this in four hours, and then giving 50 cc. every four hours until the blood culture is negative. Nilsson[14] noted no difference in the therapeutic value of serum manufactured for human use and veterinary serum. Because of favorable results obtained by the use of organic arsenicals,[15, 16] these drugs may be employed as adjuvants to serum or as the main therapeutic agent if serum is not obtainable. The organic arsenicals, if used, should be given in one or two large doses. Small doses do not appear to have sufficient therapeutic effect to justify their use, and their cumulative effect is probably as dangerous as that of one or two large doses.

Case Report

A 40-year-old man was admitted to St. Joseph's Hospital on September 7, 1940, with an anthrax pustule on the flexor surface of the left forearm at the level of the junction between the middle and the distal thirds of the radius. A dark-brown eschar, measuring about 2.5 cm. in diameter, was surrounded by a number of vesicles filled with hemorrhagic fluid. About 3 cm. from the eschar were two bullae filled with a brown serous fluid. The skin from the level of the middle part of the humerus to the fingers was markedly edematous, indurated, doughy and pink. The tissue in the edematous area revealed discrete pinpoint areas of hemorrhagic infiltration. Except at the time of injury, there was no complaint of pain, although the patient noted a feeling of pressure and numbness. The infected arm was about twice the size of the right arm. The axillary nodes were tremendously enlarged. The temperature was 100.2°F., the pulse 92, and the respirations 24; the white-cell count was 20,400.

The patient stated that 9 days before admission, while handling wool, he stuck a burr into the anterior surface of the left forearm. Except for slight itching in the inoculated area, there was no disturbance. The following day, he noticed a "burning sensation" and the appearance of an "area of redness." On the same day, a "pimple" developed, from which he squeezed pus. On the 3rd day, the surrounding skin began to "swell, and became hard and stiff." The swelling continued, and the hand and arm became so large that the patient sought medical advice.

At the time of admission, stained smears from the contents of the pustule and vesicles showed staphylococci but no evidence of anthrax bacilli. Agar plates inoculated directly from the pustule revealed staphylococci. A blood culture showed growth, and stained smears revealed gram-positive bacilli having the characteristic appearance of anthrax bacilli. Stained smears were again made from the contents of the vesicles; they showed anthrax bacilli. An emulsion was made, using one of the colonies on a blood-agar plate. Twenty-nine hours after the injection of 0.5 cc. of the preparation into the subcutaneous tissue of the abdominal wall of a guinea pig, the animal died. Necropsy showed a gelatinous infiltration of the superficial structures of the abdominal wall. The liver and spleen were markedly enlarged and congested. The suprarenal glands consisted of two large hemorrhagic cysts.

The kidneys showed pinpoint hemorrhagic areas. A stained smear of the free peritoneal fluid revealed the presence of anthrax bacilli, as did smears from the liver, spleen and suprarenal glands. Anthrax bacilli were grown in pure culture from the heart's blood and the suprarenal glands.

The patient was put to bed with the arm at absolute rest and in an elevated position. Staphylococci having been reported on the smears and culture, 2 gm. of sulfathiazole was administered at once. This amount was repeated in 4 hours, and then 1 gm. every 4 hours was given. Twenty-four hours later, when the blood culture revealed anthrax bacilli, antianthrax serum was administered; 150 cc. of serum was injected intravenously, and 50 cc. was injected subcutaneously at several points about the lesion. Six hours later, 200 cc. of serum was given intravenously, and 50 cc. was injected deep into the muscular tissue in the upper arm at the level of the upper and middle thirds of the humerus and again around the lesion. Six hours later, another 200 cc. of serum was injected intravenously. The amount was then cut to 100 cc. intravenously and 50 cc. about the lesion, given at 6-hour intervals. A total of 850 cc. of serum was administered in a period of 18 hours. Blood cultures taken the following morning, 17 hours after the beginning of antianthrax serum therapy, revealed the presence of anthrax bacilli. Twenty-four hours later, cultures and smears were reported negative for bacilli. Thus, after 14 days of illness, 24 hours of antianthrax serum and sulfathiazole therapy produced negative smears and cultures. Pending the report of another blood culture, 100 cc. of serum was given every 8 hours. Blood cultures and smears from the site of inoculation continued to be negative. Serum was omitted, but sulfathiazole was continued until September 16. The sulfathiazole level on September 15 was 5.1 mg. per 100 cc. A total of 1150 cc. of serum was given.

During the illness, the temperature varied from 98 to 102°F. The white-cell count fluctuated between 20,300 and 10,400. The blood smears and blood chemical findings were not remarkable. It is to be noted that 12 hours after the deep intramuscular circular injection of serum in the upper arm, a linear demarcated area of pinpoint hemorrhages was seen. Neither swelling nor dermal changes occurred above the injected area. Except for slight serum sickness, which was controlled by calcium gluconate and epinephrine in oil, the patient made an uneventful recovery and was discharged cured.

This case demonstrates the beneficial effects of massive doses of antianthrax serum. However, since staphylococci were found on smears and in cultures, sulfathiazole was administered. In view of the facts that the most beneficial effects are obtained when antianthrax serum is used early, that late administration, even in massive doses, shows discouraging results and that, in this case, the serum was given effectively late in the course of the disease, the question is, Does sulfathiazole act synergistically with serum? In-vitro experiments performed on six different occasions showed that anthrax bacilli grew less readily on plates containing sulfathiazole powder and antianthrax serum than they did on plates containing sulfathiazole or serum alone. Although these experiments do not prove conclusively that sulfathiazole is an effective adjuvant, the results serve as a basis for further experiment.

Summary

The modern diagnosis and treatment of anthrax are discussed.

A case successfully treated with antianthrax serum and sulfathiazole is reported, and the possible value of the latter as an effective therapeutic adjuvant is considered.

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MEDICAL PROGRESS

THORACIC SURGERY

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BRONCHIAL ADENOMA

IN last year's review,[1] attention was called to the discrepancies that may occur in end-result statistics purporting to deal with primary bronchogenic carcinoma by different interpretations of the nature of the so-called "bronchial adenoma." There are now sufficient definitive characteristics on record to permit the recognition of this tumor both clinically and pathologically in nearly every case. Only occasionally is the small fragment of tissue obtained by bronchoscopic biopsy inadequate for an unmistakable histologic diagnosis. If certain controversial aspects of these tumors are avoided, there seems to be general agreement on the following characteristics:

Slow rate of growth. Not infrequently, there is direct proof from symptoms, radiography or bronchoscopic biopsy that the tumor has been present five years or longer and still remains small and sharply circumscribed.

Early age incidence. Approximately four fifths of the reported cases occur before the age of forty.

Relatively equal sex incidence. Several series of cases indicate that the tumor occurs in females with a frequency as great as or even greater than that in males.

Low metastasizing power. Metastases have only rarely been demonstrated in adjacent lymph nodes. The evidence indicating remote hematogenous metastases is not as yet clearly definitive. Generalized metastases comparable to those commonly observed in the terminal stages of cancer of other organs have not been described.

Surgical resectability. Technical difficulties in surgical resection are almost invariably due to the inflammatory complications of prolonged bronchial obstruction rather than to mediastinal invasion or lymph-node involvement.

Low operative mortality rate. The operative mortality rate is approximately 10 per cent, depending somewhat on whether a lobectomy or a total pneumonectomy is performed.

Permanent cures by resection. Patients surviving resection appear to be permanently cured. No case has been reported in which the patient died of recurrence or metastases.

Central location. With rare exceptions, the tumors occur in the large bronchi near the hilum and are thus within easy reach of the bronchoscope.

Typical histologic pattern. Small uniformly sized cells are arranged in columns, showing few if any mitoses. Bone formation is not unusual.

If tumors with these characteristics are set aside, it will be found that other bronchogenic tumors portray more typically the picture of carcinoma as established for other organs. Such tumors grow rapidly, varying with the degree of cellular differentiation; the greatest age incidence is from forty to sixty years; the sex incidence is predominantly male (as in cancers of the lip, tongue and stomach); metastases are frequent, again varying with cellular type; surgical resectability, the mortality rate of resection and the permanence of surgical cure are discouraging, favorable results depending on early diagnosis, and recurrence or metastases taking a heavy toll among patients surviving operation; the site of the primary lesion may be peripheral or central, varying in relation to the type of cell; the histologic patterns are those of epidermoid carcinoma, adenocarcinoma or undifferentiated carcinoma, as found in other organs.[2]

Controversy regarding these tumors is concerned with definitions and interpretations rather than with the observational facts listed above. It is not the purpose of this review to defend any thesis relative to the nature of bronchial adenomas, or to criticize any proposed system of nomenclature. One point, however, seems sufficiently obvious: if cases of so-called "adenoma" are mingled with cases of other bronchogenic tumors in a study from which conclusions are drawn regarding prognosis or therapy, this fact should be clearly stated by the author. Compilations of the results of surgical therapy[3] or of x-ray therapy[4] are of doubtful value, or may be positively misleading if this simple rule is not observed.

All articles in this series will be published in book form; the current volume is *Medical Progress: Annual, 1940* (Springfield, Illinois: Charles C Thomas Company, 1941. $4.00).

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War Injuries of the Chest

Statistics

A recent personal communication relative to chest injuries in the present war stated that the mortality in chest cases received from the evacuation following the Battle of France was only 5 per cent, or equal to only one third the mortality in the South African campaign of 1900, which was the lowest hitherto recorded, and one fifth that in World War I.

It is important to realize just what such figures mean, and the following quotation from a book[5] published in 1877 is so pertinent that further comment is unnecessary.

> *Apparent increase in ratios of mortality in certain wounds.* — The following fact serves to illustrate the difference which there may appear to be in the relative fatality of certain wounds under hospital treatment, owing to differences in the distances of the hospitals from the places of fighting. During the Crimean War the percentage of fatality of chest wounds, all kinds of chest wounds being taken together, in the French military hospitals was 30.7 per cent. This ratio of mortality was almost the same as it was in the hospitals of the British army. In the French hospitals during the Italian campaign of 1859, however, the mortality was only 18.9 per cent. The altered rate of mortality shown in the hospital returns of the latter war cannot be said to have been due to any diversity in the nature of the wounds inflicted in the field, nor in that of the treatment adopted in the hospitals; it was obviously attributable to the fact that the field-hospitals in the Crimea were close to the places where the wounds were received, while in the Italian War they were situated for the most part at a considerable distance from them. In the Crimea, the patients were received into the ambulances shortly after their wounds had been inflicted; in Italy, owing to the largeness of the number of wounded resulting from the principal battles, and the consequent difficulties of transport, together with the distance of the hospitals, many of the severer cases did not live long enough to be admitted into hospital for treatment, — they died on the field of action itself. The chest wounds among the "killed in action" in Italy were increased in number; the deaths among those treated in hospital were, in proportion, lessened in number. The absolute mortality from wounds of the chest was probably very similar in both the Italian and Crimean wars.

Body Armor

> Full on his breast the Trojan arrow fell
> But harmless bounded from the plated steel.
>
> Pope: *Translation of the Iliad.*

Body armor is used by police officers and agents of the Federal Bureau of Investigation in certain specialized situations. It furnishes good protection from missiles up to velocities of 1000 feet per second. Many attempts have been made to devise a practical body armor for modern warfare.[6] Obviously, no body armor with a weight that is supportable can be effective against high-velocity direct axial hits.

There seems to be little doubt that body armor can be used to advantage in specialized situations of modern warfare. The high fatality of chest and abdominal injuries, the frequency of injury from projectiles of low velocity and the transportation of troops by motorized equipment rather than by foot all argue for its use. It seems probable that any appreciable reduction in the mortality rate of chest injuries is apter to be achieved by preventive measures of this type than by more efficient methods of evacuation of the wounded.

Blast Injuries

Longmore[5] in 1877 referred to injuries resulting from the "disturbance by actual molecular agitation produced by the violent impulse of the air," and to "vibratory disturbance of the internal organs near to the surface on which the impact of the projected gas is directly received." He described a terrific explosion of magazines of gunpowder and munitions of war in the French siege park and the adjoining English siege-train enclosure on November 15, 1855, as follows:

> There were large numbers who, though not struck by any solid projectile, nor visibly wounded by the impulsive force of the explosion, were yet more or less seriously contused and injured by it. Some suffered from rupture of superficial blood-vessels of the air-passages and lungs, others from mischief to the organ of hearing. Especially prevalent, however, was shock to the general nervous system, and there could be little doubt, from observation, that this was not merely the result of panic but was largely due to actual physical concussion and contusion.

Blast injury is being discussed as a new entity in the present war, probably because of the frequency of its occurrence as a result of bombing from the air. Sutherland[7] describes the blast wave as a spherical shell of compressed air immediately surrounding the explosion. In the compression part of the wave, pressures up to 200 atmospheres have been observed in bomb explosions. The elasticity of compressed air tends to make it recover its original state, and in this way it tends to overshoot its mark so that although the outside shell is compressed a shell of rarefied air is left behind.

The wave of compressed air travels at an enormous speed (with a 60-pound charge, the velocity at thirty feet from the source has been recorded as 1500 feet per second). The duration of the wave of high pressure at any one point is approximately 0.006 second, and of the following phase of low pressure, 0.03 second, varying with the charge and the distance from the source.[8]

The impact of this wave on the human body causes death without signs of external injury. Sometimes there is a small amount of blood trickling from the nose. Capillary hemorrhage

and even laceration of the alveolar walls may be observed at autopsy. If death does not occur immediately, symptoms may appear as late as two to five days after the injury. Cough, shortness of breath and blood-stained sputum result from the hemorrhagic exudate into alveolar and interstitial tissue.

Many reports of blast injuries and experimental studies on the mechanism involved are appearing in current English journals.

Esophagus

Carcinoma

The relatively recent advance in the surgical management of carcinoma of the esophagus is pointed out by Adams.[9] Before 1934, only in the celebrated case treated by Torek did the patient survive operation by more than two years. Thirteen other technically successful cases are on record, but the patients died of recurrence or metastases.

Since 1938, successful resections on 34 patients have been compiled by correspondence with twelve American surgeons. Of these, 20 are living, and about 50 per cent of the total of sixty-eight operations were successful.

It may be stated without qualification that surgery offers a reasonable chance of arresting carcinoma of the esophagus. Palliative results may be secured when permanent arrest is impossible. The value of this achievement is more apparent if it is realized that carcinoma of the esophagus is fourth in frequency of all malignant tumors occurring in men over twenty years of age. It is surpassed in frequency only by cancer of the stomach, lung and rectum.

The procedure of esophageal resection is far from standardized, and at the moment calls for a combination of technics derived from gastrointestinal, thoracic and plastic surgery. In general, two quite distinct procedures are available, the choice depending on the level of the carcinoma. Approximately 45 per cent of the tumors are located in the lower third of the esophagus. By a transpleural approach, the diaphragm is divided, and the cardiac end of the stomach, as well as the esophagus, is mobilized. Resection of the tumor-bearing area is followed by elevation of the fundus of the stomach into the pleural cavity and anastomosis with the lower end of the divided esophagus, thus re-establishing the continuity of the digestive tract. This operation is also being successfully employed for carcinomas of the cardiac end of the stomach that previously have been considered inoperable when approached by the abdominal route because of extension up the esophagus, or that, if resectable from below, have required a total gastrectomy.

Carcinoma involving the upper two thirds of the esophagus demands a more complicated and mutilating procedure. No operation has been devised that will permit an internal reconstructive resection. The Torek procedure, with modifications, remains the only feasible one. Following the resection of the tumor-bearing area, the upper end of the esophagus is drawn out through a cervical incision and is left as an end esophagostomy. The lower end is inverted into the stomach after resection of the lymph nodes at the cardioesophageal angle. A preliminary Beck–Jianu gastrostomy brought well above the costal margin provides for feeding and may be connected with the cervical stoma by a rubber tube. Patients swallow well-chewed food quite satisfactorily through such a temporary artificial esophagus. The permanent goal, however, is the construction of a skin tube joining the cervical esophagostomy and the gastrostomy openings.

The execution of details in this complicated program has varied greatly, and further experience will undoubtedly point the way to simpler methods. At the present time, its successful application calls for versatility and judgment on the part of the surgeon, and patience as well as courage as the contribution of the patient.

Congenital Atresia

A study of 32 cases of congenital atresia of the esophagus has been presented by Lanman.[10] There was a fatal outcome in all the 30 operative cases, but it is evident that considerable progress along rational lines has been made. Lanman recommends a direct anastomosis if possible; otherwise, closure of the tracheoesophageal fistula is advised as the first step. As soon as conditions permit, the subsequent procedures are, first, the exteriorization of the upper esophageal segment and, secondly, an anterior gastrostomy. I have recently had the opportunity of examining a well-developed and well-nourished child at the University of Minnesota Hospital whom Dr. Nathaniel L. Leven has brought well into the second year of life by the latter procedure. The problems of the construction of an artificial esophagus in this child are being postponed until a suitable age.

Lanman confesses that he dreads to commit an infant to the sort of existence that this deformity entails, and I must admit that this very consideration has led me to limit my own efforts in dealing with congenital atresia to attempts at direct anastomosis. Needless to say, they have been uniformly unsuccessful, but perhaps technically progressive.

An indirect report has reached me that a successful anastomosis has been achieved at the University of Michigan by Dr. Cameron Haight. If

this report is confirmed, the score will be even for the two methods of approach.

Other anomalies of development are frequently associated with atresia of the esophagus and tracheoesophageal fistula. Lanman records in his series 7 cases of associated anomalies, which if not treated by operation were in themselves incompatible with life. In working with such infants, one not infrequently recalls the interpretation of Oliver Wendell Holmes:

> Many affections which art has to strive against might be easily shown to be vital to the well-being of society. Hydrocephalus, tabes mesenterica, and other similar maladies are natural agencies which cut off the children of races [that is, strains] that are sinking below the decent minimum which nature has established as the condition of vitality, before they reach the age of reproduction. They are really not so much diseases, as manifestations of congenital incapacity for life; the race would be ruined if art could ever learn always to preserve the individuals subject to them. We must do the best we can for them, but we ought also to know what these "diseases" mean.

Cardiospasm

The true nature of the disorder of the cardia associated with dilatation of the esophagus has been under discussion for years. Mosher[11] concluded that the fundamental lesion was not spasm but fibrosis of the terminal portion of the esophagus secondary to inflammatory changes starting in adjacent organs. The theory of achalasia of the cardiac sphincter associated with the name of Hurst is well known, but leaves the matter as a "chronic and progressive lesion of Auerbach's plexus," which is still far from satisfying.

An interesting concept has been proposed from Brazil (Etzel[12]), where the disease is said to occur with great frequency, and almost exclusively, among poor country people. Etzel is convinced that not only megaesophagus but megacolon and other associated conditions are the manifestations of a degenerative neuropathy of the intramural part of the autonomic nervous system caused by chronic and incomplete deficiency of vitamin B_1. Evidence to support this hypothesis is derived from histologic studies and animal experimentation. Citation is made of MacCarrison's work describing enteric lesions in B_1 avitaminosis, in which lesions of a degenerative type were disclosed in Auerbach's plexus.

If this hypothesis can be confirmed, it is necessary to postulate a subclinical avitaminosis producing irreversible degeneration of certain cells of the autonomic system. The known tendency for the onset of the clinical syndrome to be associated with anxiety states or psychic trauma suggests that psychosomatic channels simply provide the trigger mechanism.

The treatment of the disease is usually satisfactorily carried out by dilatation. In complicated or intractable cases, an esophagocardioplasty may be recommended. Even with complete symptomatic relief of the obstructive symptoms, the megaesophagus persists.

Bronchiectasis

Two important studies throw light on the prognosis of untreated bronchiectasis and indicate that the extension of radical surgery in the treatment of this disease has been justified by the poor outlook under other forms of therapy. Perry and King[13] issued a report based on 260 patients not treated by major operations. Of a group of 211 patients traced, 66 are known to be dead, a mortality rate of 31 per cent. Forty-one per cent of these had died within five years of the onset of symptoms. The series includes recently diagnosed cases, so that the mortality rate reflects only partly the toll that may be assumed in a late follow-up study. Seventy-eight per cent of the deaths were directly attributable to the disease.

Bradshaw et al.[14] report on 171 patients with bronchiectasis, excluding those cases attributable to foreign body or pulmonary abscess, and excluding those patients treated by major surgery or regular bronchoscopic aspirations. In these patients, the diagnosis was established between the years 1925 and 1935. Fifty-nine, or 34.5 per cent, died from bronchiectasis or its complications. The average duration of life in the dead patients from the onset of symptoms was thirteen and a half years.

The conclusion is justified that bronchiectasis is a fatal disease. Death occurs most commonly from pneumonia, and also from pulmonary abscess and gangrene, brain abscess, empyema and, rarely, hemorrhage.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27351*

PRESENTATION OF CASE

A 3½-pound newborn infant was admitted four hours after delivery because of prematurity, having been born early in the eighth month of pregnancy, by spontaneous breech delivery after seven hours of labor.

The family history was noncontributory. The mother had been well during pregnancy, and the cause of the premature delivery was not known.

At birth, the baby responded well, had a good cry, and allegedly was not cyanotic. Physical examination on entry revealed a small, fairly well-developed, moderately cyanotic infant whose temperature was 95.6°F. The respirations were regular, but there was slight dullness posteriorly, with a few scattered rales at both bases. The heart and abdomen seemed normal.

Examination of the blood showed a red-cell count of 5,850,000, and a white-cell count of 21,600 with many young cells of both the white and red series. The differential count was 40 per cent polymorphonuclears, 48 per cent lymphocytes, 5 per cent monocytes, 6 per cent eosinophils, and 1 per cent basophils, with normal platelets.

The baby was placed in a high-oxygen concentration in a heated cradle, and whenever taken out became cyanotic. Lumbar puncture on the morning after admission revealed grossly bloody fluid. During the second hospital day, respirations became irregular, the color poor, and the cry weak. Respirations ceased that afternoon, twenty-four hours after birth.

DIFFERENTIAL DIAGNOSIS

DR. THEODORE H. INGALLS: Here we have a baby born prematurely by breech extraction, weighing 3½ pounds at birth, who seemed relatively normal after delivery. Subsequently, he developed increasingly grave spells of cyanosis; dullness and rales appeared at the bases of the lungs. The patient rapidly failed and died within twenty-four hours.

The most striking symptom we have to consider is cyanosis. Cyanosis occurs when the concentration of reduced hemoglobin in the blood reaches approximately 5 gm. per 100 cc. It is well to bear in mind that cyanosis is not necessarily synonymous with anoxia of tissue cells, although of course it may be the precursor. The first possible cause of cyanosis in this case is the interference with continuous respiratory exchange, which to some degree at least is a normal happening at birth. Although some babies are never cyanotic at birth, many are quite blue, and many are absolutely livid before they take their first gasp. In this particular case, however, we are given to believe that neither the attending physician nor, subsequently, the nurses were alarmed about the baby's color.

Secondly, cyanosis can be caused by cardiac and pulmonary disturbances, such as the admixture of venous blood with arterial blood through pathologic openings associated with congenital heart disease. Except for the cyanosis, no other findings in this case draw one's attention particularly to the heart. Atelectasis may be a cause of cyanosis. However, simple atelectasis as the only cause of the failure of the lungs to expand is not a satisfactory diagnosis in my opinion. Gross abnormalities of the bronchial tree or the inspiration of such large amounts of mucus and amniotic fluid that the baby practically drowns would result in atelectasis, but one would expect the appearance of the symptoms soon after birth.

This brings me to the final and most serious cause of cyanosis — cerebral injury, which may result from asphyxia, trauma, hemorrhage, edema or a combination of these. In fact, they are usually combined. Following asphyxia, for example, the first result is congestion, then edema, then necrosis and finally hemorrhage. In this case, a very tangible suggestion that there was cerebral injury is grossly bloody lumbar puncture. Such a finding is also consistent with the fact that this baby was distinctly premature. The fragile underdeveloped tissues of the premature infant make him particularly susceptible to cerebral injury, so that he is frequently unable to withstand even the normal stress and strain and anoxemia of delivery. In this case, birth was by breech extraction, which is a distinct central-nervous-system hazard.

The symptoms of cerebral hemorrhage depend on the size, the site and the propagation of the hemorrhage. If there is slowly accumulating subarachnoid bleeding, there may be irritation of the cortex, and convulsions may ensue. If there is subtentorial bleeding, cyanosis may be the predominating symptom. In this case, a suggestion that this may have occurred is the fact that the baby's fontanelle is not mentioned as bulging. Finally, we have the findings in the chest to in-

*This case is published through the courtesy of the Children's Hospital.

terpret — dullness and rales at the bases of the lungs, which I should think represented pulmonary edema due to injury of the vagal nuclei; if both vagi are injured in the experimental animal, pulmonary edema follows consistently in the space of twelve to twenty-four hours. It is not possible to rule out pneumonia, although I am under the impression that pulmonary edema is more often misdiagnosed as terminal pneumonia than the reverse. I shall be interested in the pathological report on the lungs, however.

Finally, I think I should merely mention vitamin K deficiency in passing as being a possible, or at least a contributory, cause of bleeding, although in this case the only clinical suggestion of bleeding is the bloody lumbar puncture.

Dr. Eli C. Romberg: It would have been interesting to know whether the baby had any slowing of the pulse and of respirations, because all the premature infants who have those symptoms and intermittent cyanosis, and die within thirty-six hours, have subtentorial hemorrhage.

Dr. Harold L. Higgins: The striking thing about this case to me is that we are dealing with a premature infant, and I cannot help believing that a most likely cause of death was the inability of the child's brain to maintain normal respiration and circulation, because of immaturity. I realize that an intracranial hemorrhage was suspected, because a lumbar puncture was done; but I wonder if there was bulging of the fontanelle or other signs of increased pressure. Again, was the blood found on spinal puncture due to trauma? I should certainly suspect vitamin K deficiency as a contributing cause of the hemorrhage. My diagnosis would be prematurity, with probable cerebral hemorrhage, hemorrhagic disease of the newborn and traumatic injury at birth.

Clinical Diagnoses

Prematurity.
Cerebral hemorrhage.

Dr. Ingalls's Diagnoses

Prematurity.
Cerebral hemorrhage (? subtentorial).
Pulmonary edema (? pneumonia).

Anatomical Diagnoses

Pneumonia (pneumococcus).
Pneumococcemia.
Pulmonary edema and congestion.
Aspiration of amniotic-sac contents.
Cerebral hemorrhage, subarachnoid and subdural.
Prematurity.

Pathological Discussion

Dr. Sidney Farber*: I think the fact that so many possibilities have been mentioned in the thorough review that Dr. Ingalls gave, in addition to the fact that different suggestions have been made concerning what the infant might have had, is characteristic of the problem of the premature infant. This infant weighed 3½ pounds, and died twenty-four hours after entering the hospital. At autopsy, there was diffuse hemorrhage beneath the arachnoid and beneath the dura, and a hematoma of the scalp. So much for the cerebral difficulty. There was a very early pneumococcal pneumonia. The pneumococcus was grown from the heart's blood and the lung. Furthermore, in the lungs, there was evidence of aspiration of amniotic-sac contents and considerable congestion and edema. Fibrin thrombi were found microscopically in many organs of the body, a finding that may be explained by the pneumococcemia.

I should like to say before going farther that prematurity or immaturity of tissues alone is in my opinion an unsatisfactory cause of death or of any important disturbance in a baby weighing 3½ pounds. Although immaturity of the tissues is usually demonstrable, particularly in the brain, I believe that a baby of this weight has achieved enough maturity of development to get along perfectly well and to live without having serious symptoms referable to immaturity of tissues.

The meninges were extremely thin. The superficial vessels were congested, and there was diffuse subarachnoid hemorrhage scattered over the various brain surfaces. Some of this appeared to be quite recent and it is probable that not all the hemorrhage took place during labor. There was no collection of blood over the cerebellum or brain stem, sites of involvement that would cause much more intense cyanosis or much more difficulty with respiration than the type of hemorrhage encountered here. If we find no more cerebral hemorrhage than we found here, we must look elsewhere for a satisfactory explanation of the cyanosis. A premature baby rarely suffers from tentorial tears or rupture of the great veins of Galen. Gross tears of the dural membranes do not occur so frequently as in full-term babies.

The mode of entrance of the pneumococci was by way of the aspirated amniotic contents, which had been infected with the organisms from the birth canal of the mother.

In summary, there was diffuse cerebral hemorrhage as a part of a general birth injury. This

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was a contributory cause of death and of the cyanosis. The immediate cause of death was the pneumococcemia, which spread to the blood stream from the lungs. The pneumococcus apparently entered the lungs with aspirated amniotic-sac contents, which were infected from the birth canal of the mother.

DR. HIGGINS: One wonders about infection as the result of mouth-to-mouth insufflation.

DR. FARBER: That would be quite possible as the means of entrance of the organisms.

A PHYSICIAN: Is the white-cell count of 22,000 high?

DR. INGALLS: I should consider it moderately high for a premature infant; it did not strike me as pathognomonic of infection.

DR. HIGGINS: Do you not think, Dr. Farber, that one can get functional immaturity without pathological immaturity?

DR. FARBER: I am quite sure one could. My point, Dr. Higgins, is that, although immaturity of the respiratory center is of importance and is one of the factors that combined with others may bring about atelectasis and respiratory difficulty, immaturity alone is not explanation enough for respiratory disturbance in a premature baby.

CASE 27352

PRESENTATION OF CASE

First Admission. A forty-three-year-old American roofer entered the hospital complaining of a skin rash.

Three years before entry, he noticed a slightly raised reddish itching patch about 1 cm. in diameter on the inside of the right thigh. Since then, similar lesions had appeared all over his body, including the abdomen, back, chest and the flexor surfaces of the lower legs and arms. No lesions had ever appeared on the extensor surfaces of the arms or legs or on the head. The lesions were usually round at the onset and finally became completely irregular. Some of them developed brown crusts; others became rough, without crusts. The lesions usually lasted about three weeks and left no marks. There was no seasonal distribution. Three months before entry, a crusting lesion appeared in the right groin. This swelled considerably and then disappeared.

The family, marital and past histories were noncontributory.

Physical examination showed a well-developed and well-nourished man lying in bed without discomfort. Over the back of the neck, the flexor surfaces of the arms, and over the back and abdomen, there were slightly raised pink lesions varying from 1 to 4 cm. in diameter. Some were irregularly bordered, others sharply outlined. Many showed only slight scaling, whereas others had a grayish crust.

The temperature was 98.4°F., the pulse 80, and the respirations 20.

Examination of the urine was negative. The blood showed a red-cell count of 5,090,000 with a hemoglobin of 75 per cent, and a white-cell count of 5650 with 74 per cent polymorphonuclears.

An x-ray film of the chest showed that the right lung field was slightly less radiant than the left, and that the intercostal spaces were narrowed. There was evidence of thickening of the pleura from the apex to the diaphragm, but no definite areas of consolidation within either lung. The lung roots were slightly thickened. There were no mediastinal masses.

On the fifth day, a biopsy of one of the skin lesions was taken, and an axillary lymph node removed. The skin showed the histologic structure of mycosis fungoides, but the axillary lymph node showed only chronic inflammation. The patient was discharged on the following day, after having received x-ray treatment and one intravenous injection of arsphenamine.

Final Admission (approximately four years later). Following discharge, he was seen fairly often, receiving on the average one x-ray treatment a month. The larger lesions were fairly well controlled by this treatment. Seven months before entry, while in Maine, he developed a very large lesion on the right thigh. He went to another physician, who gave him three doses of very deep x-ray therapy, the last one about a month before entry. This treatment, according to the patient, "knocked the stuffing out of him," and he was forced to go to bed. He was very weak and had a very poor appetite. During the month before this admission, he developed slight shortness of breath, and about a week later, an abscess in the left axilla, which discharged spontaneously.

Physical examination showed an emaciated, anemic, cachectic man, with generalized, blotchy, red and pigmented, scaly, macular areas, and also several infiltrated plaques and nodules over the skin. There was generalized lymphadenopathy. The mucous membranes were pale but showed no lesions. There was slight bronchovesicular breathing over both bases, but no rales. The heart sounds were of poor quality. The blood pressure was 100 systolic, 55 diastolic. The liver edge was felt 5 cm. below the costal margin. The spleen was felt 5 cm. below the umbilicus and in the flank.

The temperature was 102°F., the pulse 110, and the respirations 20.

Examination of the urine was negative. The blood showed a red-cell count of 3,190,000 with a hemoglobin of 65 per cent, and a white-cell count of 5400 with 64 per cent polymorphonuclears. The platelets were markedly decreased. The red blood cells showed mild achromia. A blood Hinton reaction was negative. The bleeding time was 3 minutes.

An x-ray film of the chest was negative.

The patient was given fluid parenterally. The temperature remained fairly elevated, ranging between 100 and 102°F. The red-cell count dropped to 1,160,000, with a hemoglobin of 35 to 40 per cent. The white-cell count dropped to 2600 and finally to 1250. A smear showed marked achromia and variations in size and shape of the red blood cells. No platelets or young forms were seen. There was approximately 70 per cent polymorphonuclears. The patient failed rapidly, and died sixteen days after admission.

Differential Diagnosis

Dr. Walter F. Lever: A differential diagnostic discussion of the cutaneous lesions is superfluous, because the diagnosis "mycosis fungoides" had already been established by histologic examination, four years before the patient's death. In addition to the cutaneous eruption, the patient presented a generalized lymphadenopathy, a marked increase in the size of the liver and spleen, and a severe anemia, leukopenia and thrombocytopenia. The question arises whether any or all these manifestations were caused by mycosis fungoides.

According to the present prevailing view, mycosis fungoides is a form of lymphoblastoma. Under lymphoblastoma are grouped all neoplasms derived from the stem cell, which is the mother cell of both the lymphatic and the reticulum cells. The infiltrate of mycosis fungoides contains, in varying proportions, stem cells and both types of cells derived from the stem cell, so that histologic examination may show stem cells, lymphoblasts, lymphocytes, reticulum cells and monocytes. In addition to these, a variety of other cells are usually present in the infiltrate, namely granulocytes (neutrophils, as well as eosinophils) fibroblasts, fibrocytes and plasma cells, all of which are mature. The latter group of cells represents merely an inflammatory reaction provoked by the neoplastic infiltrate. This inflammatory reaction is most pronounced in the early stages of the disease, so that histologic examination at that time may show only mature cells, as found in chronic inflammation. If the neoplastic process involves mainly the lymphatic cells, mycosis fungoides may, histologically as well as clinically, resemble lymphocytic lymphoblastoma; if the infiltrate is made up mainly of reticulum cells, the histologic picture of mycosis fungoides may resemble reticulum-cell lymphoblastoma; if both types of cells are present in a more or less equal proportion, mycosis fungoides may histologically resemble Hodgkin's disease. There even exists strong evidence that mycosis fungoides is actually identical with any of the three above-mentioned types of lymphoblastoma, and that it differs from them only clinically in its tendency to primary and preponderant localization in the skin.

The classification of mycosis fungoides into the group of lymphoblastomas and the realization of its close relation to, if not identity with, lymphocytic lymphoblastoma, reticulum-cell lymphoblastoma and Hodgkin's disease explain the occasionally encountered widespread neoplastic involvement of internal organs in mycosis fungoides.

Applying this concept to the present case, I believe that histologic examination of some lymph nodes showed neoplastic changes involving either the lymphatic or the reticulum cells, or both types of cells simultaneously, whereas other lymph nodes showed only an inflammatory infiltration, just as the cutaneous lesions of mycosis fungoides may present either a neoplastic or merely an inflammatory infiltrate.

The considerable enlargement of the liver and spleen was, in my opinion, also due to an infiltration with neoplastic lymphoblastic cells. Other causes than lymphoblastoma can be ruled out fairly well. The tremendous size of the spleen readily rules out the possibility of cirrhosis of the liver, or of cardiac decompensation or amyloidosis. Furthermore, there were no collateral symptoms to substantiate these diagnoses.

There are three possible explanations of the aplastic anemia. First, the aplastic anemia may have been due to lymphoblastic involvement of the bone marrow (myelophthisis). Secondly, it may have been merely a terminal, nonspecific, toxic process caused by cachexia, and thirdly, it may have been the result of the extensive x-ray therapy. In mycosis fungoides, bone-marrow changes are fairly frequent but, as a rule, are present only in a mild degree. They occur mainly in the form of an increase in the number of stem cells and their immature derivative cells. These bone-marrow changes of mycosis fungoides may occur with or without changes in the number and appearance of the circulating blood cells. Yet, I should think that an unusually severe involvement, which in the end completely prevented the

emission of red and white blood cells into circulation, would have led to an appearance of immature blood cells during the preceding weeks. In view of the absence of qualitative changes in the peripheral blood, I am inclined to believe that the aplastic anemia was a nonspecific toxic terminal process. The extensive x-ray radiation may have been a contributory factor. I believe it will be impossible to determine how much either factor was responsible. In any case, I should expect the bone marrow to show the usual appearance in aplastic anemia (fatty, gelatinous degeneration, and microscopically almost complete absence of red cells, myelocytes and megakaryocytes).

In conclusion, I want to point out the clinical resemblance of this case of mycosis fungoides to one of Hodgkin's disease. I believe that the resemblance to Hodgkin's disease will be borne out by the histologic examination.

Clinical Diagnosis

Mycosis fungoides.

Dr. Lever's Diagnoses

Lymphoblastoma (mycosis fungoides, ? Hodgkin's disease) of the skin, lymph nodes, liver and spleen.

Aplastic anemia (terminal toxic, with x-ray irradiation possibly a contributory factor).

Anatomical Diagnoses

Mycosis fungoides.
Aplastic anemia.
Pulmonary infarction, septic, multiple, bilateral.
Infarct of spleen.
Operative wound: biopsy, left epitrochlear gland.
Arteriosclerosis, slight, aortic.

Pathological Discussion

Dr. Tracy B. Mallory: Dr. Lever's predictions proved very close to what we actually found at autopsy. The primary diagnosis had, of course, already been established histologically by the biopsy. There is, however, great variety among these cases in the type of infiltrating cells and in the degree of involvement of the internal organs. In this case, there was obvious lymphomatous involvement of a large proportion of the lymph nodes of the body and also of the spleen. The liver, however, did not contain neoplasm and showed only a mixture of fatty infiltration and central necrosis of the lobules. It was not strikingly large, weighing 1800 gm., whereas the spleen, which weighed 1100 gm., is well up to the size seen in leukemia. Dr. Lever was correct in stating that the bone marrow would not be replaced by tumor cells but would be consistent with aplastic anemia. It showed a great diminution in the hematopoietic elements of both the red-cell and white-cell series, a slight increase in the number of fat cells and a considerable degree of so-called "gelatinous degeneration." There were no other significant findings.

From the point of view of histologic classification, the neoplastic cells all showed differentiation in the direction of relatively mature lymphocytes. It would have been impossible, for instance, to distinguish sections of the spleen and lymph nodes from those of corresponding tissues from a case of lymphoid leukemia. As Dr. Lever has pointed out, this is by no means always so. Even when there is a marked generalized lymphadenopathy, one is often disappointed that a lymph node removed for biopsy shows only a nonspecific hyperplasia and nothing on which a diagnosis of lymphoma can be made. The diagnosis of the disease must usually be made from a biopsy of the skin itself. In the early stages, this is often extremely difficult, and sometimes several biopsies must be taken before the diagnosis can be established. The period of time during which the process may remain localized to the skin is often, in comparison with other lymphomatous processes, astoundingly long. Five and ten years are not uncommon figures, and intervals as long as fifteen years are sometimes observed.

I can go no farther than Dr. Lever did in explaining the cause of the aplastic anemia. It may have been some effect of the neoplastic process, or of the chronic sepsis that so often supervenes in the terminal stages of this disease when the skin tumors break down and ulcerate. Prolonged x-ray exposure can certainly produce this type of bone marrow, and in the course of years these patients often receive a formidable amount of radiation.

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PREMARITAL MEDICAL EXAMINATIONS

The Massachusetts Legislature has passed and Governor Saltonstall has signed, as noted elsewhere in this issue of the *Journal*, a premarital examination law that is as well conceived and as sound as that in any state. Great credit is due to Representative Leslie B. Cutler, of Needham, without whose whole-hearted interest and intelligent efforts this bill probably would not have passed. The Massachusetts Medical Society and other organizations worked hard in support of Mrs. Cutler.

This bill provides that no couple may receive a marriage license until they can provide the town or city clerk who issues it certificates showing that a registered practitioner of medicine has examined each party and has explained his findings to both parties. A blood test for syphilis is to be part of the examination. A law with similar provisions has been in successful operation in Virginia for nearly two years.

The wording of the bill makes it mandatory for the physician to find or exclude any "infectious disease declared by the State Department of Public Health to be dangerous to the public health." This means that all so-called "reportable diseases" must be considered. The bill in no way changes present laws in connection with the reporting to local boards of health of cases found to have any of these infections.

The law makes no provision for halting any marriage; but if its provisions are carried out conscientiously by the profession, men and women will be prevented from marrying obviously sick persons without being so informed in advance. Furthermore, if disease is found, an opportunity is provided for the treatment of the infected prospective partner and for the protection of the other after marriage.

The bill puts a very great responsibility on those members of the profession who will make these examinations, but the duty should not be shirked and overcharges should be avoided. Nominal charges for very careful examinations might well result in creating friendships that would lead to many calls for obstetric or other medical service in later years. Furthermore, such an approach would be peculiarly effective at the time when nearly all couples, no matter how wealthy they may be, feel poor because of the large expenses necessarily incurred in setting up a home.

Nearly all premarital laws in other states focus their emphasis on "blood testing" and also attempt to prevent marriage under certain conditions. The difficulty with such provisions lies in the impracticability of setting up fair and workable laws that will cover all the various contingencies. This difficulty is recognized in some states, such as New York, by providing that certain judges have the power to authorize marriages otherwise forbidden when "public policy" makes such a step seem proper. A difficulty with this is that judges as a class are not trained in medicine or public health

and must act on information secured from physicians. It seems much better to leave the whole matter to physicians, and to remove all possible compulsions beyond the determination and presentation of medically determined facts.

PROCUREMENT AND ASSIGNMENT AGENCY

ALTHOUGH over 160,000 physicians have filled out the questionnaires forwarded by the Committee on Medical Preparedness of the American Medical Association, and although these invaluable data concerning professional training, experience and availability have been transferred to punch cards at the headquarters of the association, as yet no authority has been created with the power to make use of this material.

As pointed out in *War Medicine*,* the future needs for medical personnel will probably include the following: physicians for the United States Army, Navy and Public Health Service and other governmental agencies; physicians for the local and appeal boards created under the Selective Service Act; physicians for civilian medical care and defense; physicians for aid to Britain, requested by the American Red Cross; physicians for industry; physicians for rehabilitation; and physicians for state and county medical and public-health organizations. The procurement and assignment of men to fill these positions to best advantage is a tremendous task, but one that could be properly handled if the punch-card data were utilized. For this reason, the House of Delegates, at the recent meeting of the American Medical Association, passed a resolution urging the establishment, by the United States government, of a central authority, to be known as the Procurement and Assignment Agency, to provide, if necessary, for efficient and adequate medical staffing of all governmental, civilian and industrial services. The membership of such an agency would include representatives of the civilian medical profession.

Copies of this resolution were forwarded to the various governmental heads in Washington who are concerned with the problems of medical care, and it is hoped that prompt action will be taken and that means will thus be created for the provision of a wisely selected medical personnel in any emergency.

*Editorial: A procurement and assignment agency. *War Med.* 1:536-538, 1941.

MEDICAL EPONYM

BENCE-JONES PROTEIN

A paper, "On a new substance occurring in the Urine of a patient with Mollities Ossium," was read before the Royal Society on April 22, 1847, by Henry Bence-Jones (1813–1873), physician to St. George's Hospital. This appears in the *Philosophical Transactions of the Royal Society of London* (**138**, Pt. 1: 55–62, 1848).

> On the 1st of November 1845 I received from Dr. *Watson* the following note, with a test tube containing a thick, yellow, semi-solid substance: — 'The tube contains urine of very high specific gravity; when boiled it becomes highly opake; on the addition of nitric acid it effervesces, assumes a reddish hue, becomes quite clear, but, as it cools, assumes the consistence and appearance which you see: heat reliquifies it. What is it?'
>
> A few hours afterward a specimen of the same urine, passed by a grocer forty-seven years of age, who had been out of health for thirteen months, was sent to me by Dr. *MacIntyre*. He, being in attendance on the case with Dr. *Watson*, had two days previously first observed the peculiar reactions of the urine.
>
> The specimen of urine was slightly acid; specific gravity 1.034; it contained a sediment consisting of crystalline phosphate of lime, oxalate of lime, and cylinders of fibrin. The urine became thick with heat from a deposit of phosphates, but cleared with a drop of acid. It gave no precipitate with an excess of nitric acid, unless left to stand, or unless heated and left to cool, when it became solid. This solid redissolved by heat, and again formed on cooling. Continued boiling with strong nitric acid evolved but little gas, and did not quickly hinder this reaction. Hydrochloric acid gave the same solid precipitate, soluble by heat. Strong acetic acid gave only a slight precipitate which redissolved by heat. Caustic potash and sulphate of copper gave a splendid bright blue, clear liquid, passing over when heated to claret colour. . . .
>
> January 2nd. — The patient died. The following day I saw that the bony structure of the ribs was cut with the greatest ease, and that the bodies of the vertebrae were capable of being sliced off with the knife. For an account of the structure of the bone, see a paper by Mr. *Dalrymple* in the third number of the Dublin Journal, August 1846. . . .
>
> The ultimate analysis of this substance may be represented by C_{48}, H_{37}, N_6, O_{15} or C_{40}, H_{30}, N_5, O_{12}. . . . Hence it is an oxide of albumen, and from ultimate analysis, it is the hydrated deutoxide of albumen. . . .

The peculiar characteristic of this hydrated deutoxide of albumen was its solubility in boiling water, and the precipitate with nitric acid being dissolved by heat and reformed when cold. . . .

This substance must again be looked for in acute cases of mollities ossium. The reddening of the urine on the addition of nitric acid might perhaps lead to the rediscovery of it. . . .

R. W. B.

OBITUARY

NORMAN CLYDE BAKER

1883 – 1941

Norman Clyde Baker was born on January 27, 1883, in Meredith, New Hampshire, and died suddenly at the Massachusetts General Hospital on July 31, 1941. A graduate of Jefferson Medical College in Philadelphia in 1908, he served an internship in the Rhode Island Hospital in 1909, later acting as assistant superintendent or admitting physician. From 1922–1926 he was superintendent of the Newport Hospital, Newport, Rhode Island.

He came to the Massachusetts General Hospital in 1927, as an assistant director in charge of the development of plans for the Baker Memorial. Promoted to first assistant director in 1928, he filled this responsible position until his death.

During World War I, Dr. Baker went to Halifax, Nova Scotia, in charge of the Red Cross unit sent from Providence for the relief of victims of that terrible disaster. Later he served as a captain in the Medical Corps of the United States Army. He belonged to numerous medical and hospital societies and was secretary of the Massachusetts Hospital Association from 1936 to 1940.

In 1917, Dr. Baker married Josephine Halton, a graduate of the Rhode Island Hospital School of Nursing. He is survived by his wife and one son, Norman Baker, Jr.

For several years, Dr. Baker's activities were curtailed by a circulatory weakness, but in spite of this he carried on ably his exacting duties at the hospital. Friendly, cordial, thoughtful of others, painstaking and sagacious in the investigation of hospital problems, wise in judgment and decision, Dr. Baker was an outstanding example of the efficient hospital administrator.

He was beloved by all with whom he came in contact.

N. W. F.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

Mid-Forceps Delivery Followed by Pulmonary Embolus and Death

A thirty-three-year-old primipara was delivered at term by mid-forceps, and died suddenly seventeen days after delivery, presumably of a pulmonary embolus.

The past history was irrelevant. Physical examination was negative. The patient had been seen routinely throughout her pregnancy, which had been uneventful until two weeks before delivery, when the blood pressure rose to 140 systolic and the patient showed slight albuminuria and edema. She was not hospitalized at the time, so that it is fair to infer that the toxemia was mild.

Labor started spontaneously and lasted thirty-six hours, the membranes rupturing sixteen hours before delivery. The mid-forceps operation was done because of the lack of progress. The preparation of the patient consisted in a soap-and-water scrub and the application of 4 per cent Mercurochrome. The hospital record shows that a temperature of 101°F. was reached on the second day; the temperature vacillated from 99 to 101°F. until the ninth day, when it came down to normal and remained there for forty-eight hours. From then on, it vacillated from normal to 103°F. The day before death, the temperature was 97°F. in the morning, and the patient was allowed out of bed. In the afternoon, the temperature rose to 100°F. Death was sudden.

During convalescence, the patient was given urotropin, and the hospital record states that there was tenderness in one leg.

Comment. It is interesting to note that the skin preparation at the time of delivery consisted of a soap-and-water scrub and the application of 4 per cent Mercurochrome. In many of the better hospitals, patients are now prepared for delivery with ether and 1 per cent iodine. (One per cent iodine has been proved to be as efficient as half-strength iodine and less irritating; preceded by ether, it is vastly superior to a questionable preparation consisting of a soap-and-water scrub and the application of an antiseptic like Mercurochrome, the bactericidal value of which is relatively low.) Standardization of skin prepara-

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section. Letters should be addressed to Dr. Raymond S. Titus, *Secretary*, 330 Dartmouth Street, Boston.

tions should be established. Ether and iodine have been proved bactericidally to be as good as any other preparations and better than most, and the iodine solution costs less than 10 cents a pint, a figure much lower than those of other antiseptic solutions.

This patient evidently was septic from the beginning, and it is quite likely that she was suffering from a pelvic phlebitis. The use of urotropin during the convalescence suggests that pyelitis was wrongly diagnosed. There is no excuse for allowing a patient who has had tenderness in the leg and an elevated temperature on and off for eighteen days to get out of bed. This death, which was undoubtedly caused by an embolus, and which might have been prevented by conservative treatment, was due to poor medical care.

COMMITTEE ON STATE AND NATIONAL LEGISLATION

The following is a transcript of House Bill No. 460, recently signed by Governor Saltonstall:

AN ACT further regulating the filing of notices of intention of marriage, and the delivery of certificates of such intention and the return of unused certificates.

Section 1. Chapter two hundred and seven of the General Laws is hereby amended by inserting after section twenty A, inserted by section three of chapter two hundred sixty-nine of the acts of nineteen hundred and thirty-nine, the following new section:

> Section 20B. Except as hereinafter provided, such notice of intention of marriage shall not be accepted by the clerk or registrar until he has received from each party to the intended marriage a certificate signed by a registered physician who has examined such party as hereinafter provided. If such physician, in making such examination, discovers evidence of any infectious disease declared by the state department of public health to be dangerous to the public health, he shall inform both parties of the nature of such infectious disease and of the possibilities of transmitting the same to his or her marital partner or to their children. Such examination shall include a standard serological test for syphilis and said test shall be made by a laboratory of said department or by a laboratory approved by it for such test.
>
> Such certificate shall read as follows: — I (name and address of physician), a registered physician of (city or town) in the commonwealth of Massachusetts on oath declare that on (day, month, year) I examined (name and address of party) in accordance with section twenty B of chapter two hundred and seven of the General Laws.
>
> The examination by such physician and the laboratory test shall be made not more than thirty days before the filing of the notice of marriage. Whoever fails to comply with this section shall be punished by a fine of not less than ten nor more than one hundred dollars. In extraordinary or emergency cases where the death of either party is imminent or where the female is near the termination of her pregnancy, upon the authoritative request of a minister, clergyman, priest, rabbi or attending physician, the clerk or registrar may accept such notice of intention without having received the physician's certificate hereinbefore referred to.

Section 2. Section twenty-eight of said chapter two hundred and seven, as appearing in the Tercentenary Edition, is hereby amended by striking out, in the third and in the eleventh lines, the words "six months" and inserting in place thereof, in each instance, the words, "sixty days," so as to read as follows: —

> Section 28. On or after the fifth day from the filing of notice of intention of marriage, except as otherwise provided, but not in any event later than sixty days after such filing, the clerk or registrar shall deliver to the parties a certificate signed by him, specifying the date when notice was filed with him and all facts relative to the marriage which are required by law to be ascertained and recorded, except those relative to the person by whom the marriage is to be solemnized. Such certificate shall be delivered to the minister or magistrate before whom the marriage is to be contracted, before he proceeds to solemnize the same. If such certificate is not sooner used, it shall be returned to the office issuing it within sixty days after the date when notice of intention of marriage was filed.

Section 3. Section fifty-seven of said chapter two hundred and seven, as so appearing, is hereby amended by striking out, in the second and in the fourth and fifth lines, the words "six months" and inserting in place thereof, in each instance, the words, "sixty days," so as to read as follows: —

> Section 57. Whoever performs a ceremony of marriage upon a certificate more than sixty days after the filing of the notice of intention of marriage as set forth in such certificate, and whoever having taken out such certificate and not having used it fails to return it, within sixty days after such filing, to the office issuing the same, shall be punished by a fine of not more than ten dollars.

Section 4. This act shall not affect any certificate referred to in section twenty-eight of chapter two hundred and seven of the General Laws which was lawfully issued and outstanding upon the effective date of this act.

HENRY C. MARBLE, *Chairman*

DEATH

TRACY — WILLIAM L. TRACY, M.D., of Pittsfield, died recently. He was in his sixty-seventh year.

Born in Hartland, New Brunswick, he attended Fredericton Normal School and the University of New Brunswick, and received his degree from McGill University Faculty of Medicine in 1908. He was treasurer and chief surgeon of Hillcrest Hospital, Pittsfield.

Dr. Tracy was a fellow of the Massachusetts Medical Society, the American Medical Association and the American College of Surgeons. He was president of the Pittsfield Surgeons Club and was treasurer of the Pittsfield Cancer Clinic.

His sister, two nieces and five nephews survive him.

MISCELLANY

AMERICAN MEDICAL DIRECTORY

About September 1, an information card will be sent from the headquarters office of the American Medical Association to every physician in the United States and Canada. The information secured is to be used in compiling the seventeenth edition of the *American Medical Directory*.

The directory is prepared at regular intervals in the Biographical Department of the American Medical Association. The last previous edition appeared in 1940. This volume is one of the most important contributions of the American Medical Association to the work of the medical profession in the United States; it has been especially valuable in the medical preparedness program. In it, as in no other published directory, are dependable data concerning physicians, hospitals, medical organizations and activities. The directory provides full information concerning medical colleges, specialization in the field of medical practice, memberships in special medical societies, tabulations of medical journals and medical libraries and, indeed, practically every important fact concerning the medical profession in which anyone might possibly be interested.

Before filling out the information card, physicians should read the instructions carefully. They are especially urged to state whether or not they are on extended active duty for the medical reserve corps of the United States Army and Navy. The card should be filled out and returned promptly whether or not a change has occurred in any points on which information is requested. If a change of address occurs before March 1, 1942, it should be reported at once. If any physician fails to receive a card before October 1, he should write at once to the headquarters office stating that fact, and a duplicate card will be mailed.

NOTES

Three National Scholarships, the outstanding awards to students entering Harvard Medical School this fall, were recently announced by Harvard University. The recipients are Gaston E. Blom, of Tuckahoe, New York, A.B. Colgate University '41; William F. Ketchum, of Evanston, Illinois, A.B. Harvard '41; and Frederick R. Gilmore, of Bloomsburg, Pennsylvania, A.B. Lehigh University '41. This is the fifth year of the medical school's National Scholarship plan, which is similar to that introduced in Harvard College in 1934, and subsequently adopted by the Graduate School of Arts and Sciences, the Graduate School of Design and the Graduate School of Business Administration.

In addition, medical-school scholarships and fellowships totaling $6,860, for study during the next academic year, were awarded as follows: Matthew and Mary E. Bartlett Scholarship to Harold D. Rosenbaum, of Fair Play, Kentucky; James Jackson Cabot Fellowship to Carl T. Nelson, of Jamaica Plain, Massachusetts; DeLamar student research fellowships to William E. Watts, of Seattle, Washington, Victor C. Vaughan, 3rd, of Richmond, Virginia, and Israel H. Scheinberg, of New York City; Jeffrey Richardson Fellowship to Eugene R. Sullivan, assistant in medicine, Massachusetts General Hospital; George Cheyne Shattuck Memorial Fellowship to Walter E. Knox, of McCook, Nebraska; Charles Eliot Ware Memorial Fellowship to William F. Pollock, of Santa Monica, California; John Ware Memorial Fellowship to Herbert R. Morgan, of Bell, California; Abraham A. Watson Scholarship to Stuart G. Quan, of Oakland, California; Whitman Fellowship to Henry S. Fuller, of Washington, D. C.; and Dr. William Hunter Workman Scholarship to Ping-Yang Liu, research fellow in bacteriology and immunology, Harvard Medical School.

The Board of Directors of Oglethorpe University has recently announced the opening of a school of medicine, to be located on the university campus, in the suburbs of Atlanta, Georgia. Clinical facilities will be available at the Lawson General Hospital, Grady Hospital and U. S. Veterans' Hospital No. 48. Dr. John L. Jacobs, formerly professor of bacteriology and immunology at Tufts College Medical School, has been elected vice-president of the university in charge of the scientific work of the institution and is now engaged in the organization of courses, admission of students and preparation of classes. Although the school is primarily intended for Georgia residents, anyone may apply for admission.

At the annual meeting of the Massachusetts Board of Registration in Medicine held July 11, 1941, Dr. Francis R. Mahony, of Lowell, was re-elected chairman of the Board and Dr. Stephen Rushmore, of Boston, was re-elected secretary for the ensuing year.

BOOK REVIEWS

Criminal Youth and the Borstal System. By William Healy, M.D., and Benedict S. Alper. 8°, cloth, 251 pp. New York: Commonwealth Fund, 1941. $1.50.

Since four out of five of the "graduates" of reformatories for delinquents and young criminals commit further offenses on their release, any investigation made by competent scholars on the methods of preventing this human waste ought to be welcomed not only by sociologists and criminologists but by physicians, whose fine tradition in the healing art, tested by time, might well be projected, both civically and professionally, to co-operate in the prevention of crime.

Criminal careers are mainly a problem of youth, and there has been a startling increase of criminality in the 'teen ages. More major crimes are committed at the ages of nineteen and twenty than at any other age. Previous studies have shown reformatories to be dehumanized, depersonalized and regimented — in other words, inefficient. Although improvement in these institutions is not the sole or even the chief means of prevention, anything to improve their administration would help. Accordingly, the Criminal Justice-Youth Committee of the American Law Institute commissioned Dr. Healy, a noted authority on delinquents, and Mr. Alper to make a study of the Borstal system in Great Britain, which, according to the report presented, seems to work quite well.

The authors sketch vividly the organization, personnel and day-to-day life in Borstal and similar institutions, and they give the reader an intimate account of the whole procedure from court commitment to aftercare and parole. The Commonwealth Fund is to be congratulated on the publication of this able, objective and penetrating report.

Proctology and the General Practitioner. By Frederick C. Smith, M.D., M.Sc. (med.). 8°, cloth, 466 pp., with 161 illustrations and 5 color plates. Philadelphia: F. A. Davis Company, 1941. $4.50.

This book, as the name implies, is primarily written for the general practitioner, but it can also be read with benefit by the general surgeon and the proctologist. It

may be divided into three sections: diseases of the anus and the anorectal junction; major diseases of the rectum and colon; and parasitic diseases of the entire gastrointestinal tract.

The greater part of the book is devoted to the first section. The chapters on anatomy, anorectal symptoms, history taking, methods of examination, preoperative and postoperative care, choice and methods of anesthesia, and the treatment of hemorrhoids, fistulas, fissures, acute infection and pruritus are comprehensive. Little known to the proctologist is omitted, although some proctologists will not agree with some of the author's conceptions. The most practical features of all these diseases are clearly presented, omitting all unnecessary details and controversial theoretical considerations.

The chapters on major diseases of the rectum and colon, such as cancer, diverticulitis and ulcerative colitis, are very sketchy, being mere outlines.

The chapter on parasitic diseases of the gastrointestinal tract is long and inclusive. It contains very valuable, practical information on clinical parasitology, an outstanding feature of the whole book.

The book is written in simple style, the type is large, and the illustrations are adequate. There are numerous witty expressions and rhetorical phrases to distract the reader from the technical monotony. For example, the author refers to cancer of the rectum and sigmoid as "the wild beast of the jungle, stalking silently on its victim and giving little warning of its approach until it makes its fatal strike."

The book makes a valuable addition to the library of the general practitioner.

The Mask of Sanity: An attempt to reinterpret the so-called psychopathic personality. By Hervey Cleckley, B.S., B.A. (Oxon.), M.D. 8°, cloth, 298 pp. St. Louis: The C. V. Mosby Company, 1941. $3.00.

This work is, on the whole, as journalistic as the title. Fourteen cases of psychopathic personality are cited, all of them in men, although there are actually as many women who are psychopathic inferiors as there are men. The case histories are given with great fervor and gusto, intermingled with bits of poetry. In fact, the introduction to the book is apparently an original poem, which begins:

> From chaos shaped, the Bios grows. In bone
> And viscus broods the Id. And who can say
> Whence Eros comes? Or chart his troubled way? . . .

The reviewer wonders what this has to do with the psychopathic personality.

The vivid description of the cases is followed by an analysis of the literature, together with the introduction of a new name for the psychopathic inferior, "semantic dementia," which means a dementia in the understanding of the meaning of life. If that is the disease from which the psychopathic inferior suffers, this term can be applied to most of us, and certainly to the reviewer, since so far as he knows, no one as yet has given us a real insight into the meaning of life. Psychiatry does not need any new names for old subjects, and progress is not recorded in this way.

No essentially new facts are presented. The book is not literary and fictional enough for a novel, yet it has a little too much literariness for a scientific publication. Its merits reside in the earnest interest of the author and his emphasis on the seriousness of the problem, although he has no real remedy to offer. In point of fact, no one else has.

Macleod's Physiology in Modern Medicine. Edited by Philip Bard, Ph.D. With the collaboration of Henry C. Bazett, M.D., L.R.C.P., George R. Cowgill, Ph.D., Howard J. Curtis, A.M., Ph.D., Harry Eagle, M.D., Chalmers L. Gemmill, M.D., Magnus I. Gregersen, Ph.D., Roy G. Hoskins, Ph.D., M.D., J. M. D. Olmsted, Ph.D., and Carl F. Schmidt, M.D. Ninth edition. 8°, cloth, 1256 pp., with 124 tables and 387 illustrations. St. Louis: The C. V. Mosby Company, 1941. $10.00.

The tradition of seven editions of this work under the late J. J. R. Macleod, eminent Canadian physiologist, now edited for the second time by Philip Bard, professor of physiology at Johns Hopkins University, makes it the outstanding treatise in this field. It is not intended to be a textbook in applied physiology, as the title implies. Although the original purpose of the book was to serve as a guide to the clinical application of physiology, the present editor and his collaborators, still devoting a great deal of attention to clinical material, deal with it only so far as it illustrates fundamental principles. They assume that the greatest service which physiology can render modern medicine is to continue to solve fundamental problems, even though they may yet not be of immediate practical application.

One may turn to this volume, then, not only as a rich authoritative source book, but as a pathfinder and pacemaker in modern medicine.

Handbook of Anaesthetics. (Formerly by Ross and Fairlie.) Revised by R. J. Minnitt, M.D. (Liverpool), D.A. (D.C.P. & S. Eng.). With chapters on local and spinal anesthesia by W. Quarry Wood, M.D., Ch.M., F.R.C.S.E. Fifth edition. 12°, cloth, 364 pp., with 103 illustrations. Baltimore: Williams and Wilkins Company, 1940. $4.00.

The fifth edition of this handbook covers the field of anesthesia quite completely and presents detailed information about the management of the patient under general anesthesia. The authors have done well to treat the subject, including the physiology of anesthetics, the preparation of the patient and the technics of administrations of anesthetics, so briefly and at the same time to give the reader a working knowledge of the underlying fundamentals.

The anesthetics are taken up in order, and cover inhalation, intravenous, rectal, local and spinal agents. The author places especial emphasis on ethyl chloride, chloroform, ether and nitrous oxide, since anesthetists in England still use these more toxic agents. The newer agents and technics that have been more recently introduced in that country, such as cyclopropane with carbon dioxide absorption anesthesia, vinesthene and the therapeutic use of oxygen, helium and carbon dioxide, are only briefly outlined.

In the section on local and spinal anesthesia, there is quite a discussion of local agents and technics and a very brief chapter on spinal anesthesia.

There are many cuts of apparatus, many of which are different from those used in the United States.

The clinical anesthetist and the medical student should find this book well worth reading. It gives a clear-cut picture of anesthesia in England.

NOTICES

ANNOUNCEMENT

DR. D. J. BRISTOL, JR., announces the removal of his office from 330 Dartmouth Street, to 319 Longwood Avenue, Boston.

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS, UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

CLINIC	DATE	CLINIC CONSULTANT
Haverhill	September 3	William T. Green
Lowell	September 5	Albert H. Brewster
Salem	September 8	Paul W. Hugenberger
Gardner	September 9	Mark H. Rogers
Brockton	September 11	George H. Van Gorder
Pittsfield	September 15	Frank A. Slowick
Northampton	September 17	Garry deN. Hough, Jr.
Worcester	September 19	John W. O'Meara
Fall River	September 22	Eugene A. McCarthy
Hyannis	September 23	Paul L. Norton

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly clinical conference and meeting of the staff of the New England Hospital for Women and Children will be held on Thursday, September 4, in the classroom of the nurses' residence at 7:15 p.m. Dr. Dorothy K. Scheidell, chairman, will speak on "Occult Kidney Infection."

EXAMINATIONS FOR APPOINTMENTS IN THE MEDICAL CORPS OF THE UNITED STATES NAVY

Examinations for appointment as assistant surgeon (lieutenant, junior grade), United States Navy Medical Corps, will be held at all the large naval hospitals and the Naval Medical Center, Washington, D. C., on October 6 to 9, inclusive, and January 5 to 9, 1942, inclusive.

Applicants for appointment as assistant surgeon must be citizens of the United States, more than twenty-one but less than thirty-two years of age at the time of acceptance of appointment, and graduates of Class A medical schools who have completed at least one year of intern training in a hospital approved by the Council on Medical Education and Hospitals of the American Medical Association.

Examinations for appointment as acting assistant surgeon for intern training in naval hospitals accredited for intern training by the Council on Medical Education and Hospitals of the American Medical Association will also be held on October 6 to 9, inclusive, and January 5 to 9, 1942, inclusive. Applicants for appointment as acting assistant surgeon for intern training must be citizens of the United States, more than twenty-one but less than thirty-two years of age, and members of the junior or senior classes in Class A medical schools. After twelve months of intern training, acting assistant surgeons may apply for appointment as assistant surgeons in the United States Navy.

A circular of information regarding these examinations, physical requirements, rates of pay and promotion and retirement data may be obtained from the Bureau of Medicine and Surgery, Navy Department, Washington, D. C., on request. Applications for authorization to take the examination must be received at the Bureau of Medicine and Surgery three weeks before the examination.

Medical officers of the United States Navy are encouraged to develop a specialty, and are assigned, if their interest in the specialty warrants such action, to postgraduate instruction in the large naval hospitals, the Naval Medical Center, Washington, D. C., and civilian medical centers. Some of the specialties in which medical officers may seek qualifications are: surgery, medicine, ophthalmology, otolaryngology, roentgenology, laboratory, pathology, public health, psychiatry, deep-sea diving, aviation medicine (flight surgery), gas warfare, tropical medicine, medical research and so forth.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The forty-sixth annual meeting of the American Academy of Ophthalmology and Otolaryngology will be held at the Palmer House, Chicago, October 19 to 23.

The academy's program consists of one general scientific meeting on the morning of the first day, separate programs for the two specialties on alternate afternoons and instructional courses every morning beginning Tuesday.

During the convention there will be various meetings of small groups, including the "Teacher's Section," secretaries of local eye, ear, nose and throat societies and alumni organizations.

Alternating with the scientific programs of the specialties each afternoon will be an elaborate motion-picture program. Thus when the section of ophthalmology is meeting for formal presentation of papers, motion pictures on otolaryngology will be available for those interested in that field.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING SUNDAY, AUGUST 31

WEDNESDAY, SEPTEMBER 3

*12 m. Clinicopathological conference. Children's Hospital.

THURSDAY, SEPTEMBER 4

7:15 p.m. Occult Kidney Infection. Dr. Dorothy K. Scheidell. New England Hospital for Women and Children.

*Open to the medical profession.

SEPTEMBER 1–5. American Congress of Physical Therapy. Page 997, issue of June 5.

SEPTEMBER 1–5. American Occupational Therapy Association. The Mayflower, Washington, D. C.

SEPTEMBER 5–6. New England Surgical Society. Hanover, N. H.

SEPTEMBER 16–18. Clinical Congress of the Connecticut State Medical Society. Page x, issue of July 17.

OCTOBER 8–10. American Academy of Pediatrics, Boston.

OCTOBER 13–24. 1941 Graduate Fortnight of the New York Academy of Medicine. Page 834, issue of May 8.

OCTOBER 14–17. American Public Health Association. Page 579, issue of March 27.

OCTOBER 19–23 — American Academy of Ophthalmology and Otolaryngology. Notice above.

NOVEMBER 3–7. American College of Surgeons. Page vii, issue of July 31.

JANUARY 3, 1942. American Board of Obstetrics and Gynecology. Page 1079, issue of June 19.

APRIL 20–24. American College of Physicians. Page 996, issue of June 5.